

Textbook of
Interventional Cardiology
A Global Perspective

Jaypee Brothers

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A Global Perspective

Editor-in-Chief

Samir Kapadia MD

Section Head, Invasive and Interventional Cardiology
Director, Sones Cardiac Catheterization Laboratories
Director, Interventional Cardiology Fellowship Program
Department of Cardiovascular Medicine
Cleveland Clinic, Cleveland, Ohio, USA

Global Editors

Derek Chew MD MBBS MPH FRACP

Professor, Cardiology
Flinders University
Adelaide, South Australia, Australia

Fernando Cura MD PhD

Chief, Interventional Cardiology
ICBA, Buenos Aires, Argentina
Director, Interventional Cardiology and Endovascular Therapies
Instituto Cardiovascular de Buenos Aires and Sanatorio Anchorena
Buenos Aires, Argentina

Philippe L L'Allier MD

Chief, Interventional Cardiology
Montreal Heart Institute (MHI)
Montreal, Quebec, Canada

Marco Roffi MD

Director, Interventional Cardiology, Geneva, Switzerland
Vice-Chairman of Cardiology, and Director of the Interventional Cardiology Unit
University Hospital of Geneva
Geneva, Switzerland

E Murat Tuzcu MD

Professor, Medicine
Cleveland Clinic Lerner College of Medicine
Case Western Reserve University, Cleveland, Ohio, USA
Chief Academic Officer and Chief of Cardiovascular Medicine
Cleveland Clinic Abu Dhabi
Abu Dhabi, UAE



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Jaypee Brothers Medical Publishers (P) Ltd

Headquarters

Jaypee Brothers Medical Publishers (P) Ltd
4838/24, Ansari Road, Daryaganj
New Delhi 110 002, India
Phone: +91-11-43574357
Fax: +91-11-43574314
Email: jaypee@jaypeebrothers.com

Overseas Offices

J.P. Medical Ltd
83 Victoria Street, London
SW1H 0HW (UK)
Phone: +44 20 3170 8910
Fax: +44 (0)20 3008 6180
Email: info@jpmedpub.com

Jaypee-Highlights Medical Publishers Inc
City of Knowledge, Bld. 235, 2nd Floor, Clayton
Panama City, Panama
Phone: +1 507-301-0496
Fax: +1 507-301-0499
Email: cservice@jphmedical.com

Jaypee Brothers Medical Publishers (P) Ltd
17/1-B Babar Road, Block-B, Shaymali
Mohammadpur, Dhaka-1207
Bangladesh
Mobile: +08801912003485
Email: jaypeedhaka@gmail.com

Jaypee Brothers Medical Publishers (P) Ltd
Bhotahity, Kathmandu, Nepal
Phone +977-9741283608
Email: kathmandu@jaypeebrothers.com

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Website: www.jaypeedigital.com

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Dedication

To our families for their selfless support.

To our mentors for invaluable life lessons.

To our institutions for successful partnership.

Jaypee Brothers

Contributors

Ahmed Abdel-Latif MD PhD

Assistant Professor
Department of Medicine
Division of Cardiology
University of Kentucky
Lexington VA Medical Center
Lexington, Kentucky, USA

Elsayed Abo-Salem MD

Fellow
Department of Cardiovascular Health
and Diseases
University of Cincinnati
Cincinnati, Ohio, USA

Shikhar Agarwal

Chief Fellow
Interventional Cardiology
Cleveland Clinic
Cleveland, Ohio, USA

Jung-Min Ahn MD

Assistant Professor
Department of Cardiology
University of Ulsan College of Medicine
Seoul, South Korea

Said Alsidawi MD

Cardiovascular Diseases Fellow
Department of Internal Medicine
Division of Cardiology
University of Cincinnati
Cincinnati, Ohio, USA

Ivan Anderson MD

Division of Cardiovascular Medicine
Department of Medicine
University of California
Davis Medical Center
Sacramento, California, USA

Mani Arsalan MD

Department of Heart Surgery
Bad Nauheim, Germany

Ganesh Athappan MD

Department of Interventional Cardiology
Temple University Hospital
Philadelphia, Pennsylvania, USA

Usman Baber MD

The Zena and Michael A. Wiener
Cardiovascular Institute
Icahn School of Medicine at
Mount Sinai
New York City, New York, USA

Emanuele Barbato MD PhD

Professor
Cardiovascular Center Aalst
Onze-Lieve-Vrouwziekenhuis Clinic
Aalst, Belgium

Eric R Bates MD

Professor
Department of Internal Medicine
University of Michigan
Ann Arbor, Michigan, USA

Jorge Belardi MD FSCAI FACC

Clinic and Interventional Cardiologist
Director, Division of Cardiology
Instituto Cardiovascular de Buenos
Aires
Buenos Aires, Argentina

Stefan Bertog MD

Associate Professor of Medicine
University of Minnesota
Minneapolis, Minnesota, USA
CardioVascular Center
Frankfurt, Germany

Deepak L Bhatt MD MPH FACC

FAHA FSCAI FESC

Professor of Medicine
Harvard Medical School
Executive Director of Interventional
Cardiovascular Programs
Heart and Vascular Center
Brigham and Women's Hospital
Boston, Massachusetts, USA

Alessandro Cannavale MD

Department of Radiological Sciences
Vascular and Interventional Unit
Sapienza University of Rome
Rome, Italy

P Cao MD FRCS

Professor of Vascular Surgery
University of Perugia
Perugia, Italy
Chief of Vascular Surgery
Department of Cardioscience
San Camillo-Forlanini Hospital
Rome, Italy

Tiziano Cassina MD

Department of Anesthesia and
Intensive Care
Fondazione Cardiocentro Ticino
Lugano, Switzerland

João L Cavalcante MD

Assistant Professor of Medicine
Department of Medicine/Heart and
Vascular Institute
University of Pittsburgh
Medical Center
Pittsburgh, Pennsylvania, USA

Matt Certain MD

Department of Cardiology
Southeast Georgia Health Systems
Brunswick, Georgia, USA

Derek Chew MD MBBS MPH FRACP

Professor, Cardiology
Flinders University
Adelaide, South Australia, Australia

Alaide Chieffo MD

Director of Research in
Interventional Cardiology
Interventional Cardiology Unit
San Raffaele Scientific Institute
Milano, Italy

LS Cho MD

Section Head
Preventive Cardiology
Department of Cardiovascular Medicine
Cleveland Clinic
Cleveland, Ohio, USA

Ramesh G Chokka MD

Doctor
Department of Cardiology
South Australian Health and Medical
Research Institute
Adelaide, Australia

Georgios Christodoulidis MD

Icahn School of Medicine at Mount Sinai
New York City, New York, USA

Antonio Colombo MD

Interventional Cardiologist
Department of Interventional Cardiology
San Raffaele Hospital
Milano, Italy

Francesco Costa MD

Thoraxcenter
Erasmus University Medical Center
Rotterdam, The Netherlands

Fernando Cura MD PhD

Chief, Interventional Cardiology
ICBA, Buenos Aires, Argentina
Director, Interventional Cardiology and
Endovascular Therapies
Instituto Cardiovascular de Buenos Aires
and Sanatorio Anchorena
Buenos Aires, Argentina

Bharat V Dalvi MD

Consultant Cardiologist
Glenmark Cardiac Center
Mumbai, Maharashtra, India

Gianmarco de Donato MD

Assistant Professor
Department of Vascular Surgery
University of Siena
Siena, Italy

Quentin de Hemptinne MD

Doctor
Department of Medicine
Montreal Heart Institute
University of Montreal
Montreal, Quebec, Canada

Giuseppe De Luca MD PhD

Aggregate Professor of Cardiology
Department of Cardiology
Eastern Piedmont University
Novara, Italy

Suzanne de Waha MD

University Heart Centre Lübeck
University Hospital Schleswig-
Holstein
Lübeck, Germany

Sinny Delacroix MD

Doctor
Department of Cardiology
University of Adelaide
Adelaide, Australia

Milind Desai MD

Professor of Medicine
Heart and Vascular Institute
Cleveland Clinic
Cleveland, Ohio, USA

Steffen Desch MD

Professor
University Heart Center Lübeck
Lübeck, Germany

John Diaper MD

Research Assistant
Department of Anesthesia
Geneva University Hospital
Geneva, Switzerland

J Michael DiMaio MD

Director Research
Educational Training
Department of Cardiothoracic, Surgery
Baylor Scott and White
Dallas, Texas, USA

Jean-François Dorval MD

Assistant Professor
Department of Cardiology
University of Montreal
Department of Medicine
Montreal Heart Institute
Montreal, Quebec, Canada

Ariel Duran MD

Professor
Department of Cardiology
University of Uruguay, Montevideo
School of Medicine
Montevideo, Uruguay

Andrew Craig Eisenhauer MD

Director
Central Maine Heart and Vascular Institute
Lewiston, Maine, USA

Mackram F Eleid MD

Assistant Professor of Medicine
Division of Cardiovascular
Diseases and Internal Medicine
Mayo Clinic College of Medicine
Rochester, Minnesota, USA

Christoph Ellenberger MD

Staff Anesthesiologist
Department of Anesthesiology
Pharmacology and Intensive Care
University Hospital of Geneva
Geneva, Switzerland

Stephen Ellis MD

Director of Interventional Cardiology
Senior Academic Officer
Professor of Medicine
Department of Cardiology
Cleveland Clinic
Cleveland, Ohio, USA

Wael El-Mallah MD

Assistant Professor of
Internal Medicine
Texas Tech University Health
Sciences Center
Lubbock, Texas, USA

Enas A Enas MD FACC

Executive Director
Coronary Artery Disease in
Asian Foundation
Indians (CADI) Research
Lisle, Illinois, USA

Phillip A Erwin MD PhD

Interventional Cardiology Fellow
Heart and Vascular Institute
Cleveland Clinic
Cleveland, Ohio, USA

Francesco F Faletra MD

Director of Cardiac Imaging Service
Department of Cardiology
Fondazione Cardiocentro Ticino
Lugano, Switzerland

Fabrizio Fanelli MD EBIR

Department of Radiological Sciences
Vascular and Interventional Unit
Sapienza University of Rome
Rome, Italy

Carlos M Fava MD

Staff
Department of Interventional Cardiology
Fundacion Favalaro
Hospital Universitario
Buenos Aires, Argentina

Ted Feldman MD MSCAI FACC FESC

Director
Cardiac Catheterization Laboratories
NorthShore University HealthSystem
Evanston Hospital
Evanston, Illinois, USA

Roberto Ferraresi MD

Peripheral Interventional Cath Lab
Humanitas Gavazzeni
Bergamo, Italy

C Ferrer MD

Vascular Surgeon
Department of Cardioscience
San Camillo-Forlanini Hospital
Rome, Italy

Filippo Figini MD

Department of Invasive Cardiology
San Raffaele Hospital
Milano, Italy

Jennifer Franke MD

Cardiologist
Cardiovascular Center Frankfurt
Frankfurt, Germany

Georg Fuernau MD

Department of Internal Medicine/
Cardiology
University of Leipzig-Heart Center
Leipzig, Germany

Sameer Gafoor

Medical Director
Structural Heart Disease
Swedish Heart and Vascular
Seattle, Washington, USA

Giuseppe Galzerano MD

Consultant
Department of Vascular Surgery
University Hospital of Siena
Siena, Italy

Marianna Gazzetti MD

Department of Vascular Surgery
Sapienza University of Rome
Rome, Italy

Baris Gencer MD

Fellow in Cardiology
Division of Cardiology
University Hospital of Geneva
Geneva, Switzerland

Francesco Giannini MD

Interventional Cardiology Unit
San Raffaele Scientific Institute
Milano, Italy

Giuseppe Di Gioia MD

Interventional Cardiology Research Fellow
Cardiovascular Center
OLV Hospital
Aalst, Belgium

Gennaro Giustino MD

Post-Doctoral Clinical
Research Fellow
Cardiovascular Research Institute
Icahn School of Medicine at Mount Sinai
New York City, New York, USA

Frédéric Glauser MD

Doctor
Division of Angiology
University Hospital of Geneva
Geneva, Switzerland

Sachin S Goel MD

Interventional Cardiologist
Prairie Heart Institute at
St. John's Hospital
Springfield, Illinois, USA

Andrew Goodman MD

Heart Valve Center
Heart and Vascular Institute
Cleveland Clinic
Cleveland, Ohio, USA

William A Gray MD

Associate Professor of Medicine
Director, Endovascular Intervention
Department of Interventional
Cardiology
Columbia University Medical Center
New York City, New York, USA

Chris Greenman MD

Cardiology Fellow
Division of Cardiology
University of Washington
Seattle, Washington, USA

Giulio Guagliumi MD

Department of Cardiovascular
Ospedale Papa Giovanni XXIII
Bergamo, Italy

Hitinder Gurm MD

Associate Professor
Department of Interventional Cardiology
University of Michigan Health System
Ann Arbor, Michigan, USA

Tarek Helmy MD FACC FSCAI

Professor of Cardiology
Director
J Gerard Mudd Cardiac
Catheterization Laboratory
St. Louis School of Medicine
St. Louis, Missouri, USA

Luisa Heuer MD

Cardiovascular Center Frankfurt
Frankfurt, Germany

Ziyad M Hijazi MD MPH MSCAI FACC

Professor of Pediatrics
Department of Pediatrics
Weill Cornell Medical College
New York City, New York, USA
Sidra Medical and Research Center
Doha, Qatar

Iiona Hofmann MD

Cardiovascular Center Frankfurt
Frankfurt, Germany

Benjamin Honton MD

Department of Interventional Cardiology
Clinique Pasteur
Toulouse, France

L Nelson Hopkins MD

Distinguished Professor of Neurosurgery
Professor of Radiology
University at Buffalo, State University of
New York
School of Medicine and Biomedical
Sciences
Buffalo, New York, USA

Samir Kapadia MD

Section Head, Invasive and
Interventional Cardiology
Director, Sonos Cardiac Catheterization
Laboratories
Director, Interventional Cardiology
Fellowship Program
Department of Cardiovascular Medicine
Cleveland Clinic, Cleveland, Ohio, USA

Vishal Kapur MD

The Center for Interventional Vascular
Therapy, NewYork-Presbyterian Hospital
Columbia University Medical Center
New York City, New York, USA

Prashanth Katrapati MD

Fellow, Division of Cardiovascular Medicine
Department of Internal Medicine
University of Michigan
Ann Arbor, Michigan, USA

Elvin Kedhi MD PhD

Isala Hartcentrum
Isala Klinieken
Zwolle, The Netherlands

Morton J Kern MD

Professor of Medicine
University of California, Irvine
Orange, California, USA
Chief of Medicine
VA Hospital Long Beach
Long Beach, California, USA

Spencer King MD

Professor of Medicine Emeritus
Emory University School of Medicine
Atlanta, Georgia, USA

Keinichi Komukai MD

Department of Interventional Cardiology
Azienda Ospedaliera Papa
Giovanni XXIII
Bergamo, Italy

Lukas Kreienbühl MD

Division of Anesthesiology
University Hospital Geneva
Geneva, Switzerland

Amar Krishnaswamy MD

Associate Program Director
Interventional Cardiology
Cleveland Clinic
Cleveland, Ohio, USA

**Dharam J Kumbhani MD SM MRCP FACC
FAHA FSCAI**

Assistant Professor
Department of Internal Medicine
Division of Cardiology
University of Texas Southwestern
Medical School
Dallas, Texas, USA

Rony Lahoud MD

Cardiovascular Medicine Fellow
Heart and Vascular Institute
Cleveland Clinic
Cleveland, Ohio, USA

Philippe L L'Allier MD

Chief, Interventional Cardiology
Montreal Heart Institute (MHI)
Montreal, Quebec, Canada

John M Lasala MD PhD

Professor of Medicine
Director of Structural Heart Disease
Program
Department of Cardiology
Washington University School of
Medicine
Barnes-Jewish Hospital
St. Louis, Missouri, USA

Azeem Latib MB BCH

Doctor
Interventional Cardiology
EMO-GCM Centro Cuore Columbus
San Raffaele Scientific Institute
Milano, Italy

Pil Hyung Lee MD

Professor
Department of Cardiology
ASAN Medical Center
University of Ulsan College of Medicine
Seoul, South Korea

Thierry Lefèvre MD

Interventional Cardiologist
Institut Cardiovasculaire Paris Sud
Paris, France

J Leipsic MD

Associate Professor of Radiology and
Cardiology
Research Chair, Advanced Cardiac Imaging
University of British Columbia
Vancouver, British Columbia, Canada

Thibault Lhermusier MD

Washington Hospital Center
Washington, DC, USA

Marc Licker MD

Professor
Department of Anesthesia,
Pharmacology, and Intensive Care
University Hospital of Geneva
Geneva, Switzerland

A Michael Lincoff MD

Director, Cleveland Clinic Coordinating
Center for Clinical Research
Professor of Medicine Cleveland Clinic
Lerner College of Medicine Cleveland
Department of Cardiovascular Medicine
Cleveland Clinic
Ohio, USA

William Lombardi MD

Clinical Associate Professor
Department of Medicine
University of Washington
Seattle, Washington, USA

Roberto Lorenzoni MD FESC

Chief
Peripheral Interventional Program
Cardiovascular Interventional Unit
San Luca Hospital
Lucca, Tuscany, Italy

A Lorido MD

Vascular Surgeon
Department of Cardioscience
San Camillo-Forlanini Hospital
Rome, Italy

Junyang Lou MD PhD

Interventional Cardiologist
Southern Oregon Cardiology
Medford, Oregon, USA

Michael J Mack MD

Chairman
Heart Hospital Baylor Plano
Medical Director
Cardiovascular Surgery
Department of Cardiovascular
Surgery
Baylor Scott and White Health
Plano, Texas, USA

Giulia Mazzitelli MD
 Department of Vascular Surgery
 Sapienza University of Rome
 Rome, Italy

Gordon McLennan MD FSIR
 Professor
 Departments of Radiology and
 Biomedical Engineering
 Cleveland Clinic
 Cleveland, Ohio, USA

Roxana Mehran MD
 Professor of Medicine (Cardiology)
 Department of Interventional
 Cardiology
 Health Evidence and Policy Director of
 Interventional Cardiovascular Research
 and Clinical Trials
 The Zena and Michael A. Wiener
 Cardiovascular Institute
 Mount Sinai Medical Center
 New York City, New York, USA

Mariagnese Mele MD
 Department of Vascular Surgery
 Sapienza University of Rome
 Rome, Italy

Oscar A Mendiz MD
 Chief
 Department of Interventional Cardiology
 Fundacion Favalaro Hospital
 Universitario
 Buenos Aires, Argentina

John C Messenger MD
 Professor of Medicine
 Department of Medicine
 Division of Cardiology
 University of Colorado
 School of Medicine
 Aurora, Colorado, USA

M Militello PharmD
 Cardiology Pharmacy Specialist
 Department of Pharmacy
 Cleveland Clinic
 Cleveland, Ohio, USA

Erich Minar MD
 Professor
 Department of Angiology
 Medical University of Vienna
 Vienna, Austria

Tiziano Moccetti MD
 Professor
 Department of Cardiology
 Fondazione Cardiocentro Ticino
 Lugano, Switzerland

Maxim Mokin MD PhD
 Assistant Professor
 Department of Neurosurgery and
 Brain Repair
 University of South Florida
 Tampa, Florida, USA

David J Moliterno MD
 Jack M Gill Chair and Professor
 Department of Internal Medicine
 University of Kentucky
 Lexington, Kentucky, USA

Debabrata Mukherjee MD
 Chairman and Professor
 Department of Internal Medicine
 Texas Tech University Health Sciences
 Center
 El Paso, Texas, USA

C Naoum MBBS
 Cardiac Imaging Fellow
 St. Paul's Hospital
 Vancouver, British Columbia, Canada

Andres Navarro MD
 Interventional Cardiology and
 Endovascular Therapies
 Department of Cardiology
 Hospital de Los Valles
 Quito, Ecuador

Stephane Noble MD
 Department of Specialties of Medicine
 Division of Cardiology
 University Hospital of Geneva
 Geneva, Switzerland

Gerard O'Sullivan MD FSR
 Doctor
 Department of Radiology
 University College Hospital
 Galway, Ireland

Sameer Pancholy MD FACC FSCAI
 Associate Professor of Medicine
 Department of Cardiology
 Commonwealth Medical College
 Scranton, Pennsylvania, USA

Vasileios F Panoulas MD MRCP PhD
 NIHR Clinical Lecturer
 Faculty of Medicine
 Imperial College London
 London, United Kingdom

Seung-Jung Park MD
 Professor
 Department of Interventional
 Cardiology
 University of Ulsan College of Medicine
 Seoul, South Korea

Elena Pasotti MD
 Department of Cardiology
 Fondazione Cardiocentro Ticino
 Lugano, Switzerland

Manesh R Patel MD
 Associate Professor of Medicine
 Director Interventional Cardiology
 Duke University
 Durham, North Carolina, USA

Parag Patel MD
 Advanced Cardiac Imaging Fellow
 Department of Cardiovascular Medicine
 Cleveland Clinic
 Cleveland, Ohio, USA

Tejas Patel MD DM FACC FSCAI
 Chairman and Chief Interventional
 Cardiologist
 Department of Cardiology
 Apex Heart Institute
 Ahmedabad, Gujarat, India

Giovanni Pedrazzini MD
 Department of Cardiology
 Fondazione Cardiocentro Ticino
 Lugano, Switzerland

Femi Philip MD
 Associate Professor of Medicine
 University of California
 Davis Medical Center
 Sacramento, California, USA

Janine Pöss MD
 Doctor
 Department of Cardiology
 University Heart Centre Lübeck
 Lübeck, Germany

Roshan Prakash MBBS MRCP FRACP
Clinical Fellow
Department of Cardiology
University of British Columbia
Vancouver, British Columbia, Canada

Lourdes R Prieto MD
Associate Professor
Department of Pediatric Cardiology
Director, Pediatric and Congenital
Cardiac Catheterization Laboratory
Case Western Reserve University
Cleveland Clinic Lerner College of Medicine
Cleveland, Ohio, USA

Pritesh Punjabi MD DM
Interventional Fellow
Department of Cardiology
Rambam Medical Center
Haifa, Israel

Rishi Puri MBBS PhD
Doctor
Department of Cardiovascular Medicine
Cleveland Clinic
Cleveland, Ohio, USA

Mohammad Qasim Raza MD
Clinical Research Fellow
Department of Cardiovascular Medicine
Cleveland Clinic
Cleveland, Ohio, USA

Grant W Reed MD
Fellow
Department of Cardiovascular Medicine
Cleveland Clinic
Cleveland, Ohio, USA

Markus Reinartz MD
Cardiovascular Center Frankfurt
Frankfurt, Germany

Charanjit S Rihal MD MBA
Professor of Medicine
Division of Cardiovascular Diseases
Mayo Clinic
Rochester, Minnesota, USA

Marco Roffi MD
Director, Interventional Cardiology
Geneva, Switzerland
Vice-Chairman of Cardiology, and
Director of the Interventional
Cardiology Unit, University Hospital of
Geneva, Geneva, Switzerland

Jason H Rogers MD
Professor
Division of Cardiovascular Medicine
University of California
Davis Medical Center
Sacramento, California, USA

Ariel Roguin MD PhD
Professor
Department of Cardiology
Rambam Medical Center
Haifa, Israel

Eric E Roselli MD
Staff Surgeon
Department of Cardiothoracic Surgery
Cleveland Clinic
Cleveland, Ohio, USA

Mark Sands MD
Section Head
Interventional Radiology
Cleveland Clinic
Cleveland, Ohio, USA

Mariangela Santoni MD
Department of Radiological Sciences
Sapienza University of Rome
Rome, Italy

Antoine Sauguet MD
Department of Interventional
Cardiology
Clinique Pasteur
Toulouse, France

Paul Schoenhagen MD
Associate Professor
Department of Radiology
Cleveland Clinic
Cleveland, Ohio, USA

Ziad Sergie MD MBA
Cardiology Fellow
Cardiovascular CT/MRI
Division of Cardiology, Icahn School of
Medicine at Mount Sinai
New York City, New York, USA

Carlo Setacci MD
Full Professor
Department of Medicine, Surgery, and
Neuroscience
University of Siena
Siena, Italy

Francesco Setacci MD
Professor
Department of Vascular Surgery
Sapienza University of Rome
Rome, Italy

Arnold H Seto MD MPA
Assistant Clinical Professor
Chief, Cardiology
Long Beach Veterans Affairs Medical
Center
Long Beach, California, USA

Rohan Shah
Medical Student
Duke University School of Medicine
Durham, North Carolina, USA

Sanjay Shah MD DM
Director
Department of Cardiology
Apex Heart Institute
Ahmedabad, Gujarat, India

Samin K Sharma MD
Professor of Medicine
Department of Cardiology
Mount Sinai Hospital
New York City, New York, USA

Satyavan Sharma MD DM FACC
Professor and Head
Department of Cardiology
Bombay Hospital Institute of Medical
Sciences
Mumbai, Maharashtra, India

Mehdi H Shishehbor MD
Director
Endovascular Interventions
The Heart and Vascular Institute
Cleveland Clinic
Cleveland, Ohio, USA

Samuel L Sidharta MBBS BMedSC
Research Fellow
Cardiovascular Investigation Unit
Department of Cardiovascular Services
Royal Adelaide Hospital
Adelaide, South Australia

Horst Sievert MD
Professor
Cardiovascular Center Frankfurt
Frankfurt, Germany

Vasile Sirbu MD

Medical Doctor
Department of Cardiovascular Medicine
Azienda Ospedaliera Papa Giovanni
XXIII Hospital
Bergamo, Italy

Ernest Spitzer MD

Cardiologist
Department of Interventional Cardiology
Thoraxcenter, Erasmus Medical Center
Rotterdam, The Netherlands

James Spratt MD

Forth Valley Acute Hospitals
Larbert, Scotland

Eugenio Stabile MD

Division of Cardiology
Department of Advanced Biomedical
Sciences
Federico II University
Naples, Italy

Barbara E Stähli MD

Montreal Heart Institute
University of Montreal
Montreal, Quebec, Canada

S Steiner MD MSc

Senior Physician
Department of Interventional
Angiology
Medical Faculty
Leipzig University
Leipzig, Germany

Gregg W Stone MD

Professor of Medicine
Department of Medicine
Division of Interventional Cardiology
Columbia University Medical Center
Cardiovascular Research Foundation
New York City, New York, USA

Patcharapong Suntharos MD

Department of Pediatric Cardiology
Cleveland Clinic Children's Hospital
Cleveland, Ohio, USA

Hussam Suradi MD FACC ABVM

Assistant Professor of
Internal Medicine and Pediatrics
Department of Cardiology, Rush University
Chicago, Illinois, USA

Jean-Claude Tardif MD

Professor of Medicine
Director, Research Center
Department of Medicine
Montreal Heart Institute
University of Montreal
Montreal, Quebec, Canada

Holger Thiele MD

Professor of Internal Medicine and
Cardiology
Medical Clinic II (Cardiology, Angiology,
Intensive Care Medicine)
University Heart Center Lübeck
Lübeck, Germany

Michael P Thomas MD

Clinical Lecturer
Department of Cardiovascular Medicine
University of Michigan
Ann Arbor, Michigan, USA

Dustin Thompson MD

Associate Staff
Interventional Radiology
Cleveland Clinic
Cleveland, Ohio, USA

Matthew I Tomey MD

Fellow
The Zena and Michael A. Wiener
Cardiovascular Institute
Icahn School of Medicine at
Mount Sinai
New York City, New York, USA

Guillermo Torre-Amione MD PhD

Professor
Department of Cardiology
Tecnologico de Monterrey
Monterrey, Mexico
The Methodist Hospital
Houston, Texas, USA

Gabor Toth MD

Staff Member Interventional Cardiologist
University Heart Centre Graz
Medical University Graz
Graz, Austria

Alejandro R Treviño MD

Cardiology Fellow
Department of Cardiology
Tecnologico de Monterrey
Monterrey, Mexico

E Murat Tuzcu MD

Professor, Medicine
Cleveland Clinic Lerner College of Medicine
Case Western Reserve University
Cleveland, Ohio, USA
Chief Academic Officer and Chief of
Cardiovascular Medicine
Cleveland Clinic Abu Dhabi
Abu Dhabi, UAE

Marco Valgimigli MD PhD

Professor
Department of Cardiology
Director of Clinical Research
University of Bern
Inselspital, Bern, Switzerland

Javier A Valle MD

Fellow
Division of Cardiology
University of Colorado School of
Medicine
Aurora, Colorado, USA

Arnoud WJ van't Hof MD PhD

Department of Cardiology
Isala Hospital
Zwolle, The Netherlands

Jos C van den Berg MD PhD

Associate Professor of Radiology
University of Bern
Bern, Switzerland
Head of Service of Interventional
Radiology
Ospedale Regionale di Lugano
Sede Civico
Lugano, Switzerland

Eliseo Vano PhD

Professor of Medical Physics
Department of Radiology
Complutense University
Madrid, Spain

Laura Vaskelyte MD

Cardiovascular Center Frankfurt
Frankfurt, Germany

Andres F Vasquez MD

Division of Cardiology
Washington University School of
Medicine
St Louis, Missouri, USA

Alessandro Vivacqua MD
Cardiovascular Attending
Beaumont Hospital
Royal Oak, Michigan, USA

Ron Waksman MD
Associate Director
Interventional Cardiology
Director
Cardiovascular Research and
Advanced Education
MedStar Heart and Vascular Institute
MedStar Washington Hospital Center
Washington, DC, USA

SA Wayangankar MD MPH
Interventional Cardiology
Robert and Suzanne Tomsich
Department of Cardiovascular Medicine
Cleveland Clinic
Cleveland, Ohio, USA

Patrick L Whitlow MD
Department of Cardiovascular Medicine
Cleveland Clinic
Cleveland, Ohio, USA

Stephan Windecker MD
Professor and Chairman
Department of Cardiology
Bern University Hospital
Bern, Switzerland

Stephen G Worthley MBBS PhD
Professor
Department of Cardiology
University of Adelaide
Adelaide, Australia

Steven Yakubov MD FACC
Medical Chief, Structural Heart Disease
OhioHealth Heart and Vascular Physicians
Riverside Methodist Hospital
Columbus, Ohio, USA

Jennifer Yu MD
Mount Sinai Medical Center
Cardiovascular Institute
New York City, New York, USA

Thomas Zeller MD
Professor
Department of Angiology
Universitäts-Herzzentrum Freiburg—
Bad Krozingen
Bad Krozingen, Germany

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Foreword

When I was a fellow learning to do coronary angioplasty from Richard Myler and Simon Stertz, I had the privilege of meeting Andreas Gruentzig. By 1984, when we got to have a chat at one of his Emory University teaching courses, he was already regarded as the father of interventional cardiology. It took considerable courage in 1977 for him to be the first doctor to thread a balloon into the coronary artery to open the channel and relieve the patient's angina—and it worked. But those were the early days of the field, when all we had were rigid balloons, we had no idea of the right drugs (and doses) to have on board, and there were many patients who suffered significant complications. In spite of that, Gruentzig was an inspirational force with extraordinary charisma and infectious enthusiasm. His persona certainly helped propel the field forward.

Fast forward to the 1990s when I moved from the University of Michigan, running the catheter lab, to Cleveland Clinic, and the opposite happened to me. Over the ensuing years I had the rarefied opportunity to work with so many über-talented fellows—and especially closely with the interventional fellows for we were connected by caring for patients together, doing procedures, and engaged in research projects. This was a heady and exciting time when we were testing new drugs like abciximab, bivalirudin, clopidogrel—and new devices including stents and atherectomy. Everything was seemingly new; we had so much to learn. The group of fellows took it all on with unmatched eagerness and work ethic. Each one demonstrated early in their careers that they truly fulfilled the proverbial “triple threat” of excellence in patient care, education (teaching each other and me) and research. Every fellow wrote and published multiple papers, and many of these were in leading journals with significant impact for how interventional cardiology would subsequently be practiced. They are now leaders in the field in many countries all over the world, including Australia, New Zealand, Canada, Israel, Italy, Argentina, Korea, Switzerland, and throughout the United States.

The fellows did more than all these things. They were the pivotal inspirational force to me. Throughout our years and the chance to work together, they provided intellectual stimulation because their curiosity was limitless. And their desire to make an impact in the field that they had chosen to be their life work was unbridled. All of this was admixed with humor and fun, although it seems hard now to remember how we were somehow able to weave that in.

So it was no surprise that many years later this group of former fellows—now leaders—would come to me with the plan to write and edit a new textbook on interventional cardiology. It would be one that would feature a global perspective, since collectively this group surely had that. Once again it surfaced: the interest and willingness to take time from their busy daily professional lives and families to work assiduously on developing this information resource.

I am immensely proud of this “fellow” group, now representing many of the go to gurus in the field, for having put this wonderful textbook together along with so many other experts in the field from all over the world. This is a group of some of the finest individuals, doctors, educators, and researchers I have ever had the privilege of working with in my career. We have lasting friendships and I always relish the chance to get to visit with them, not just to reminisce about a rarefied time when there was such unique and accelerated progress in our field, but also to exchange ideas on how it can still advance. When we worked together interventional coronary procedures were quite unpredictable, with not infrequent dissections, thrombosis, and abrupt closures. Hardly were there any noncoronary structural cardiac interventions at the time, but now there are so many diverse ones with left-sided valves and beyond. So interventional cardiology grew up when we had the chance to work together. It is hardly recognizable from what it was in the 1990s.

Predictably, this textbook captures *all* of the ways it works now. And the father of the interventional cardiology—Andreas—would be thrilled to know that four decades later he spawned multiple generations of exemplary physician leaders. They will be relentless in striving to make the field better for patients. Their work to put this remarkable resource together exemplifies the same passion and spirit as when we all worked together—without question, a time I shall never forget.

Eric J Topol

Professor of Genomics
The Scripps Research Institute
Director, Scripps Translational Science Institute
La Jolla, California, USA

Preface

The field of interventional cardiology is growing rapidly across the globe, with advances in devices, techniques, imaging, and collaboration with surgical subspecialties. Technological advances and their adoption is quite variable across different continents. We, all editors of this textbook, although we practice in different continents, bring a unifying perspective from training at the Cleveland Clinic in the United States. We thought that bringing thought leaders from different continents to write a textbook would provide a unique textbook that can be practical and applicable all around the globe.

Interventional cardiology has three major foci for clinical practice and research, including coronary, peripheral, and structural interventions. The book is organized to address important disease states and interventions in each of these fronts. Each chapter is clinically focused and provides important technical pearls where necessary. Devices and their use are discussed including those available in different parts of the world. Despite being clinically oriented, the book is evidence-based, with tables and figures to provide organized presentation of available data. Each chapter also highlights important gaps in our understanding for future investigations. Case studies are provided with chapters to organize thought processes and procedural steps for practical guidance.

Authors who are world-renowned thought leaders in their fields and editors with their diverse but unifying background provide depth and consistency in the book that is unique. The production of the book with tables and illustrations of exquisite quality makes the book easy to read and comprehend. Tremendous effort has gone behind the scenes to make this project a success with outstanding contributions from all authors. Perspective on history and evolution of each field also provides a unique insight from stalwarts in the field who have lived through the advances.

We are hopeful that the book will be a great resource for fellows in training, physicians in practice eager to stay updated, and for researchers interested in understanding clinically important knowledge gaps.

Samir Kapadia MD

Section Head, Invasive and Interventional Cardiology
Director, Sones Cardiac Catheterization Laboratories
Director, Interventional Cardiology Fellowship Program
Department of Cardiovascular Medicine
Cleveland Clinic, Cleveland, Ohio, USA


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- Video 64.1:** Subaortic membrane is noted in the 3-chamber (top left) and coronal views (top right). Red arrows indicate subvalvular obstruction which creates flow turbulence and spin dephasing artifact. Note on the short-axis view (bottom center) the tunneled-shape format of these complex subaortic membranes.
- Video 64.2:** CMR imaging of paravalvular leak (PVL) post-TAVR in patient who received balloon-expandable 26 mm Edwards Sapien bioprosthesis. There are 2 distinct PVL jets noted (PVL 1 and PVL 2).
- Video 64.3:** CMR imaging of paravalvular leak (PVL) post-TAVR in patient who received self-expandable 31 mm Medtronic Core Valve bioprosthesis. There are again 2 distinct PVL jets noted.
- Video 71.1:** Four-chamber view of 2-dimensional echocardiogram on an 85-year-old man with patent ductus arteriosus reveals trivial tricuspid valve regurgitation by color Doppler and biatrial enlargement.
- Video 71.2:** Four-chamber view of 2-dimensional echocardiogram in the same patient as Video 71.1 shows severe left atrial and moderate right atrial enlargement.
- Video 71.3:** Angiogram in the aortic arch in straight lateral projection reveals a large patent ductus arteriosus (PDA) with dense opacification of the main pulmonary artery. Note, significant calcification along the contour of the PDA.
- Video 71.4:** Angiogram in the descending aorta after closure with a 10/8 Amplatzer Duct Occluder I device demonstrates the device in good position with some residual flow through the device, as is frequently seen immediately after deployment and typically disappears within hours.
- Video 71.5:** Angiogram in the descending aorta in lateral projection demonstrates a small, elongated (type E) patent ductus arteriosus (PDA). The PDA minimal diameter measured about 2 mm. Note a 6-Fr Goodale-Lubin catheter advanced from the pulmonary artery completely occludes the PDA.
- Video 71.6:** Angiogram in the descending aorta post coil occlusion of the ductus arteriosus in lateral projection shows coil across the ductus with approximately 3 coil loops in the aorta and 1 loop in the pulmonary artery. There is no residual shunt and no evidence of aortic obstruction.
- Video 71.7:** Angiogram in the descending aorta in lateral projection demonstrates a type “D” patent ductus arteriosus with two areas of narrowing, one adjacent to the ampulla and the other at the junction with the main pulmonary artery, measuring 3 and 2 mm in diameter, respectively.
- Video 71.8:** Angiogram in the descending aorta during deployment of a 5 × 4 mm Nit-Occlud patent ductus arteriosus (PDA) coil shows the coil is well seated between the two constrictions of the PDA with residual flow seen through the device. Note, the coil on the pulmonary end has not yet been advanced out of the delivery catheter.
- Video 71.9:** Angiogram in the descending aorta after releasing the Nit-Occlud patent ductus arteriosus coil shows the coil in proper position with 1 last loop in the pulmonary artery. Note, residual flow immediately after deployment commonly observed with this device. However, in the vast majority of cases, the ductus is completely occluded within 24–48 hours after deployment.
- Video 71.10:** Angiogram in the descending aorta in lateral projection demonstrates small, conical type A patent ductus arteriosus. The minimal diameter measured approximately 2 mm with the ductal ampulla measuring 4.5 mm.
- Video 71.11:** Angiogram in the descending aorta in lateral projection during placement of a 5/4 Amplatzer Duct Occluder device with the device still attached to the delivery cable shows the device in proper position with a trivial residual shunt. There is no evidence of aortic arch obstruction.
- Video 71.12:** Hand injection in the same patient as Video 71.11 performed via the delivery sheath in the main pulmonary artery prior to device release demonstrates no left pulmonary artery obstruction.
- Video 71.13:** Angiogram in the descending aorta after the Amplatzer Duct Occluder device has been released reveals device in good position and no residual shunt through the device.

- Video 71.14:** Two-dimensional echocardiogram suprasternal notch view of a 67-year-old woman with severe discrete coarctation of the aorta demonstrates flow acceleration by color Doppler and continuous flow across the aortic isthmus in systole and diastole.
- Video 71.15:** Rotational angiography in the transverse aortic arch displays the three-dimensional anatomy of a severe, native coarctation of the aorta in this 67-year-old patient.
- Videos 71.16A and B:** (A) Angiogram in the proximal transverse aortic arch performed in 34° left anterior oblique and 13° caudal angulation demonstrates severe coarctation of aorta. Note, optimal angiographic projections are chosen from the three-dimensional rotational angiogram shown in Video 71.15 obtained during the same procedure. (B) Same angiogram in straight lateral projection.
- Video 71.17:** Deployment of a Cheatham-Platinum covered stent pre-mounted on an 18 mm Balloon-In-Balloon catheter.
- Videos 71.18A and B:** (A) Angiogram in the transverse aortic arch in left anterior oblique/caudal angulation post stent placement demonstrates relief of the coarctation. The stent is well positioned with no evidence of aortic wall injury. (B) Same angiogram in straight lateral projection.
- Video 71.19:** Selective coronary angiogram of the RCA in 22 degree RAO and 15° cranial angulation reveals dilated RCA with moderate size coronary artery fistula draining to the SVC/RA junction. There are multiple tiny coronary artery branches arising from the fistula. (RA: Right atrium; RAO: Right anterior oblique; RCA: Right coronary artery; SVC: Superior vena cava).
- Videos 71.20A and B:** (A) Selective coronary angiogram in the fistula after coil deployment reveals occlusion of the main fistulous branch with some residual flow through some smaller branches. (B) Same angiogram in 72° left anterior oblique and 6° cranial angulation.
- Video 71.21:** Selective angiogram in the CAF arising from the RCA in the same patient as Figures 71.6A to D in straight lateral projection 3 years after initial coil occlusion reveals significant residual flow into the SVC/RA junction. (CAF: Coronary artery fistula; RA: Right atrium; RCA: Right coronary artery; SVC: Superior vena cava).
- Videos 71.22A and B:** (A) Selective right coronary artery (RCA) angiogram with temporary balloon occlusion of the fistula with a 6 mm balloon shows no residual flow through the fistula and no compromise to normal RCA flow. A small coronary artery branch is visualized proximal to the balloon. No ischemic changes were seen indicating that the fistula could be closed at that site. (B) Straight lateral projection of the same balloon occlusion angiogram.
- Videos 71.23A and B:** (A) Selective right coronary artery (RCA) angiogram after closure of the fistula with a 6 mm Amplatzer Vascular Plug II in the same patient as Figures 71.6A to D show the device in good position with no residual flow and excellent filling of the entire RCA with no impingement by the device. A very small coronary artery branch arising from the very proximal fistula is still patent. (B) Straight lateral projection of the same angiogram.
- Video 71.24:** Selective right coronary artery (RCA) angiogram of an asymptomatic 55-year-old patient shows a markedly dilated RCA fistula with a very convoluted course and multiple loops as it courses toward the coronary sinus. The site of drainage is the proximal coronary sinus. The proximal fistula measured approximately 15 mm. There appears to be a significant risk of thrombotic complications following closure of this type of fistula particularly in older patients. Therefore, no intervention was performed in this asymptomatic patient.
- Video 71.25:** Selective left coronary angiogram performed via a co-axial system using a 4-Fr glide catheter and a 7-Fr JL4 coronary guide catheter demonstrates a long, tortuous small to moderate sized coronary artery fistula arising from the left main coronary artery coursing anteriorly across the right ventricular outflow tract and draining into the main pulmonary artery.
- Video 71.26:** In the same patient as Video 71.25 a 0.018" Tracker infusion catheter was advanced to the distal coronary artery fistula via the 4-Fr glide catheter. Angiogram confirms the catheter course and position. A 0.018", 3/2 mm diameter Tornado coil (Cook Incorporated, Bloomington, IN) is then deployed to occlude the fistula.
- Video 71.27:** After deployment of 8 coils, selective left coronary angiogram shows complete closure of the fistula. The left main coronary artery, left anterior descending, and circumflex branches appear normal and unobstructed.
- Videos 71.28A and B:** (A) Selective right coronary artery (RCA) angiogram in straight anteroposterior projection reveals a dilated proximal RCA with a large, tortuous coronary artery fistula arising very proximally. The fistula initially courses anteriorly then curves posteriorly and enters an aneurysmal sac. The distal portion of the sac tapers into a smaller caliber before entering the right atrium near the superior vena cava junction. (B) Straight lateral projection of the same angiogram.

- Video 71.29:** In the same patient as Video 71.28 a guidewire rail was created to prepare for device placement from the venous side by advancing a 0.035" Wholey wire through the fistula and snaring it with a 15 mm snare.
- Videos 71.30A and B:** (A) Selective right coronary angiogram in slight right anterior oblique and cranial projection during deployment of a 6/4 Amplatzer Duct Occluder device demonstrates the device well positioned in the fistula without causing stenosis or compression of the right coronary artery. (B) Same angiogram in straight lateral projection.
- Video 71.31:** Selective right coronary angiogram repeated in different angiographic projections to be certain there is no coronary artery compression. The device was then released.
- Videos 71.32A and B:** (A) Selective right coronary angiogram after releasing the device demonstrates ideal position of the device in the proximal portion of the fistula without compression of the proximal right coronary artery. There is trivial flow through the device into the distal part of the fistula. (B) Same angiogram in straight lateral projection.
- Video 71.33:** Selective left coronary angiogram in a patient with recurrent coronary artery fistula after surgical ligation of a distal fistula from the circumflex coronary artery shows a severely dilated proximal left main coronary artery continuing into a long tunnel-like fistula, which opens into the left ventricle underneath the mitral valve annulus. The fistula has proximal and distal stenosis. The left anterior descending coronary artery originates proximal to the fistula.
- Video 71.34:** Selective left coronary artery angiogram in the same patient as Video 71.33 with temporary occlusion of the fistula with a 12 mm low-pressure balloon inflated across the distal stenotic segment. This angiogram was performed to evaluate for the presence of normal coronary artery branches coming off the fistulous segment prior to closure. Small collateral vessels from the left anterior descending coronary artery supplying the posterolateral segment of the left ventricle were seen not connected to the fistula. Hemodynamics, electrocardiogram and regional wall motion by transesophageal echocardiogram were monitored during balloon occlusion with no ischemic changes documented.
- Video 71.35:** A 10-mm Amplatzer Septal Occluder (ASO) device was used to occlude the distal portion of this fistula. Selective left coronary artery angiogram during ASO device deployment shows the device in proper position with some residual flow across the device.
- Video 71.36:** Because of the risk of thrombus propagating into the left main coronary artery, closure at the origin of the fistula was also performed. This selective coronary angiogram was performed during deployment of an Amplatzer Vascular Plug in the proximal fistula after distal closure with the Amplatzer Septal Occluder device.
- Videos 71.37A and B:** (A) Final selective left coronary artery angiogram after deployment of both devices reveals minimal flow across the proximal segment into the distal portion of the fistula. The left anterior descending coronary artery flow is not compromised. (B) Lateral projection of the same angiogram.
- Video 78.1:** Right upper pulmonary vein angiogram demonstrating large left-to-right shunting at the atrial level.
- Video 78.2:** Second defect noted anteriorly while sizing balloon is inflated.
- Video 78.3:** 8 and 11 French Amplatzer TorqVue delivery systems positioned across the posterior and anterior atrial septal defects, respectively.
- Video 78.4:** Deployment of first device (18 mm ASO) in the posteriorly located defect.
- Video 78.5:** Deployment of left atrial disk.
- Video 78.6:** Deployment of device across septum.
- Video 78.7:** Deployment of second device (28 mm ASO) in anterior defect.
- Video 78.8:** Cobra deformity corrected with device manipulation.
- Video 78.9:** Deployment of left atrial disk.
- Video 78.10:** Deployment of device across septum.
- Video 78.11:** Devices released.
- Video 78.12:** Devices released.
- Video 78.13:** RA angiogram demonstrating no residual shunting across the atrial septum.
- Video 78.14:** Moderate-severe paravalvular leak in anterolateral location.
- Video 78.15:** Defect diameter measuring 3.5 mm (arrow).
- Video 78.16:** Transapical access using 7 French MUOM sheath. Left ventriculogram showing moderate-severe MR (+3) with left atrial dilatation.

- Video 78.17:** Paravalvular defect crossed using 0.35" Terumo wire and Glidecath.
- Video 78.18:** Deployment of 6 mm Amplatzer muscular VSD device across defect.
- Video 78.19:** Deployment of 6 mm Amplatzer muscular VSD device across defect.
- Video 78.20:** Left ventriculogram post-intervention with trivial residual leak across mitral prosthesis.
- Video 78.21:** Trivial residual leak across mitral prosthesis.
- Video 78.22:** 6 mm Amplatzer muscular VSD device (arrow).
- Video 78.23:** Two-dimensional video of TTE.
- Video 78.24:** Color Doppler video of TTE.
- Video 78.25:** Short axis view of transthoracic echocardiography.
- Video 78.26:** Modified parasternal view of transthoracic echocardiography.
- Video 78.27:** Visualization of a 15 × 19 mm ostium secundum ASD with adequate tissue rims around the defect.
- Video 78.28:** TEE image showing the 18-mm Amplatzer Septal Occluder device successfully implanted in the ostium secundum atrial septal defect.
- Video 78.29:** TEE images in short axis view, preclosure.
- Video 78.30:** TEE images in modified parasternal view, preclosure.
- Video 78.31:** TEE images in short axis view, 3 months postclosure.
- Video 78.32:** TEE images in modified parasternal view, 3 months postclosure.
- Video 78.33:** TEE images in four-chamber view, preclosure.
- Video 78.34:** TEE images in four-chamber view, preclosure.
- Video 78.35:** TEE images in four-chamber view, 3 months postclosure.
- Video 78.36:** TEE images in four-chamber view, 3 months postclosure.
- Video 78.37:** Superior rim (arrow) in TEE.
- Video 78.38:** Four-chamber view showing transthoracic echocardiography at three months.
- Video 78.39:** Angiographic confirmation of adequate balloon positioning.
- Video 78.40:** Injection of diluted angiographic contrast in the target septal branch through the central lumen of the balloon catheter to evaluate its myocardial distribution and confirm seal.
- Video 78.41:** Injection of diluted echographic contrast in the first septal branch through the central lumen of the balloon catheter with enhancement of the septo-basal area at the point of proximal mitral-septal contact.
- Video 78.42:** Final angiographic result after injection of 2mL of ethanol in the target vessel: note the occluded first septal perforator artery and the normal anterograde flow in the LAD (final angiography performed after a 10-minute observation period before balloon deflation).
- Video 78.43:** Left coronary artery in RAO with caudal angulation.
- Video 78.44:** Left coronary artery in RAO with cranial angulation.
- Video 78.45:** Coronary angiography: RCA.
- Video 78.46:** Angiographic confirmation of adequate balloon positioning.
- Video 78.47:** Diluted radiographic contrast injection in the target septal branch to evaluate its myocardial distribution and adequate balloon seal.
- Video 78.48:** Contrast enhancement of the basal septum.
- Video 78.49:** Alcohol septal ablation: alcohol injection. Note the hyperechogenic aspect of ethanol in the basal septum.
- Video 78.50:** Transesophageal echocardiography.
- Video 78.51:** 3D echo view of wire traversing the posteriorly located perimitral prosthetic defect.
- Video 78.52:** Placement of two AVPII plugs.
- Video 78.53:** Trivial residual paravalvular regurgitation.
- Video 78.54:** Transthoracic echocardiography.
- Video 78.55:** Transthoracic echocardiography.
- Video 78.56:** RAO view of stiff wire crossing the anteriorly located aortic defect below the sinotubular junction.
- Video 78.57:** 4F Glide catheter following wire across defect.
- Video 78.58:** 4 mm AVP 4 device was placed, significantly reducing degree of regurgitation.
- Video 78.59:** Outcome: one 4 mm AVP4 plug was placed.
- Video 78.60:** Outcome: only trivial residual paravalvular regurgitation was left.

- Video 78.61:** Transseptal puncture in inferior and posterior septal position. Transseptal access (8 Fr Medtronic system) and visualization of LAA with 4Fr pigtail injecting 9 cc of contrast at 9 cc/second.
- Video 78.62:** Exchange over Amplatz Extra Stiff wire for Watchman sheath. TEE 23 mm and Fluoro 24 mm means choice of Watchman 27 mm occluder.
- Video 78.63:** Outcome is adequate compression, no significant leaks. Device was released. Dual antiplatelet therapy, aspirin for 6 months, and clopidogrel for 3 months. TEE follow up at 1, 6, and 12 months. No embolic events at 2 years.
- Video 78.64:** RAO cranial.
- Video 78.65:** RAO caudal.
- Video 78.66:** Choice of 22 mm Amplatzer Cardiac Plug (St. Jude Medical) device based on 18.5 mm measurement of landing zone by TEE (requires a 10 Fr sheath).
- Video 78.67:** Lobe placement and mild compression without “strawberry” oversizing.
- Video 78.68:** Final release position.
- Video 78.69:** TEE at 60 degree view shows good result with no peri-device flow. Device released with no safety issues. Discharged on aspirin for 6 months and clopidogrel for 3 months. TEE follow up at 1, 6 months, and annually. No evidence of TIA/stroke/thromboembolism at 2-year follow-up.
- Video 78.70:** Echo confirmed severe aortic stenosis.
- Video 78.71:** Echo confirmed severe aortic stenosis.
- Video 78.72:** Angiographic check of the femoral access before remove the sheath shows iliac rupture.
- Video 78.73:** Angiographic check of the femoral access before remove the sheath shows iliac rupture.
- Video 78.74:** Endovascular repair of the iliac rupture.
- Video 78.75:** Echo confirmed severe aortic stenosis.
- Video 78.76:** Echo confirmed severe aortic stenosis.

CHAPTER 88

Clinical Cases

EDITORIAL COMMENT

This chapter provides clinical cases to help illustrate the previous chapters. The cases are contributed by chapter authors and presented in a concise manner with angiograms. The value of these chapters best realized with careful review of video clips of different angiograms. Clinical learning points are clearly outlined for each case. In reality the learning is even more expansive as these are real cases presented by thought leaders who are expert physicians taking care of these patients.

■ CASE 1: TREATMENT OF LESION IN RCA WITH A DRUG-ELUTING STENT

Georgios Christodoulidis, Usman Baber, Roxana Mehran

History

- A 78-year-old male with known history of coronary artery disease presented to our center with substernal chest pain and associated dyspnea on minimal exertion over the last few days.
- Symptoms were relieved with rest and by the use of sublingual nitroglycerin.
- *Past medical history:* Dyslipidemia, insulin dependent diabetes, chronic kidney disease, known coronary artery disease, and systolic heart failure with an ejection fraction of 30%.
- *Surgical history:* Biventricular Pacing Implantable Defibrillator.
- *Medications:* Insulin, simvastatin, aspirin, enalapril, carvedilol, furosemide.
- *Family history:* No premature coronary disease.
- *Social history:* No smoking, no alcohol use.

Basic Investigations

- Physical examination was unremarkable.
- Admission EKG showed biventricular pacing.
- CBC was remarkable for anemia with hemoglobin of 9.6 g/dL (normal range 11.7–15.0 g/dL).
- Metabolic panel was remarkable for elevated creatinine of 2.8 mg/dL (normal range 0.6–1.4 mg/dL).
- Cardiac enzymes were mildly elevated with a peak troponin of 2.4 ng/mL (normal range 0.0–0.5 ng/mL).
- Patient was diagnosed with non ST segment elevation myocardial infarction and coronary angiography was planned.

Risk Stratification

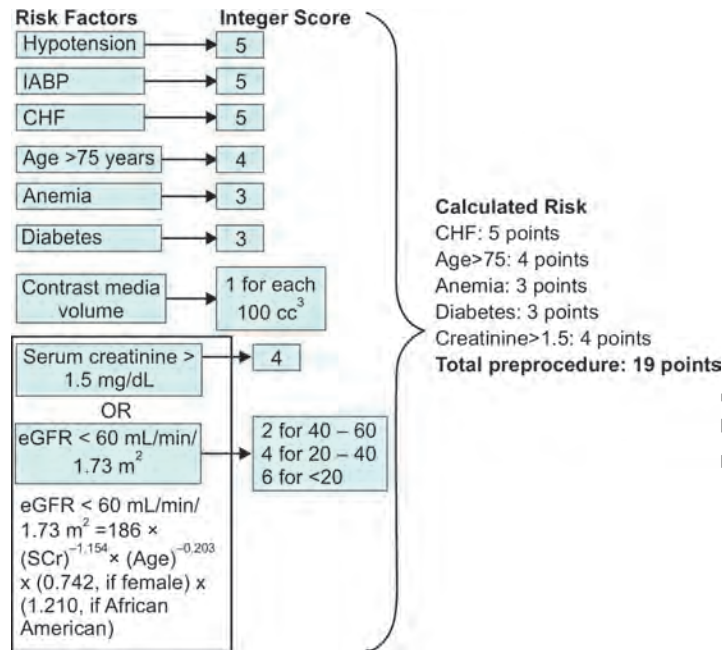


Fig. 88.1: Risk stratification.

Source: Mehran R, Aymong ED, Nikolsky E, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. *J Am Coll Cardiol.* 2004;44:1393-9.

Intervention (Management)

- Projected risk for contrast-induced nephropathy (CIN) was very high at 57% and accordingly the risk for dialysis exceeded 12%.
- Considering the high risk-features of the patient as well as his risk for overhydration, the decision was made to use the investigational device RenalGuard.
- A Foley catheter was placed, and 1 hour prior to the procedure, a 250 mL bolus of 0.9% normal saline (NS) was given, and concomitantly, furosemide at an intravenous bolus dose of 0.5 mg/kg was administered.
- Additional doses of furosemide were given whenever urine output fell below 300 mL/h.
- Contrast media given during the procedure was as low as possible (125 cc of iopamidol).
- RenalGuard therapy continued throughout the procedure, and for 4 hours afterward.

Outcomes

- Coronary angiography revealed a 95% lesion in the right coronary artery (RCA) that was treated successfully with a drug-eluting stent.
- Follow-up metabolic profile during his hospitalization did not reveal any creatinine elevation.
- Patient was discharged in a stable state of health 3 days later.
- On discharge, patient's creatinine was at his baseline of 2.6 mg/dL.

Learning Points

- Stratification for CIN risk is of utmost importance in patients undergoing angiography.
- The RenalGuard device is an investigational device which balances fluid administration rate with second-to-second furosemide induced diuresis.
- This device has been shown to decrease the risk for CIN compared to standard hydration protocols in patients at high risk for CIN.
- Additionally, RenalGuard protects patient from overhydration and pulmonary edema.

CASE 2: IN-STENT RESTENOSIS OF LEFT CIRCUMFLEX

Georgios Christodoulidis, Usman Baber, Roxana Mehran

History

- An 82-year-old female with known history of stable coronary artery disease presented to our center with worsening angina symptoms over the last month.
- She denied any symptoms at rest.
- *Past medical history:* Hypertension, dyslipidemia, diabetes, chronic kidney disease, known coronary artery disease, systolic heart failure with an ejection fraction of 38%.
- *Surgical history:* Coronary artery bypass grafting.
- *Medications:* Metformin, furosemide, atorvastatin, aspirin, clopidogrel, enalapril, carvedilol, isosorbide mononitrate, ranolazine.
- *Family history:* No premature coronary disease.
- *Social history:* Former smoker, no alcohol use.

Basic Investigations

- Physical examination was only remarkable for dry mucous membranes.
- Admission EKG showed nonspecific ST and T waves changes.
- CBC was remarkable for a hemoglobin of 16 mg/dL (normal range 11.7–15.0 g/dL), possibly as a result of hemoconcentration.
- Metabolic panel was remarkable for elevated creatinine of 2.4 mg/dL (normal range 0.6–1.4 mg/dL).
- Cardiac enzymes were negative $\times 3$.
- A coronary angiogram was planned for further evaluation.

Risk Stratification

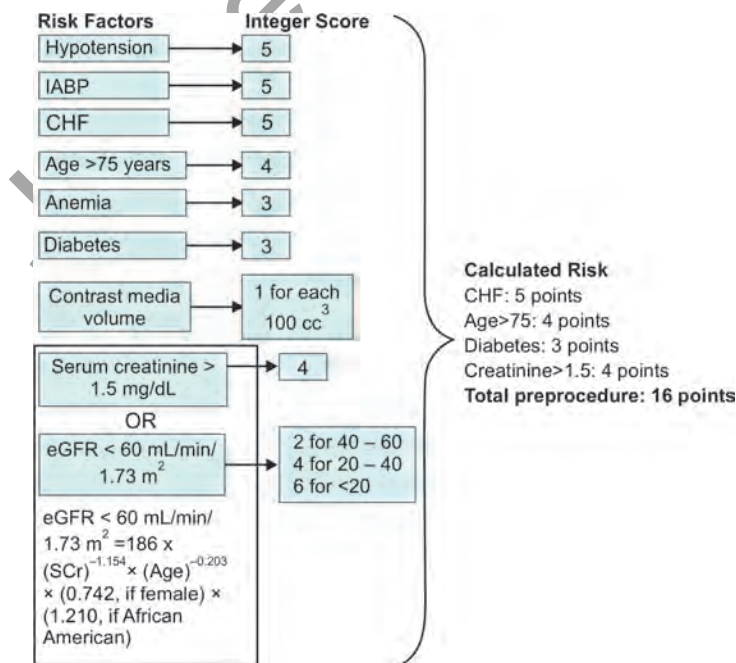


Fig. 88.2: Risk stratification.

Source: Mehran R, Aymong ED, Nikolsky E, et al. A simple risk score for prediction of contrast-induced nephropathy after percutaneous coronary intervention: development and initial validation. J Am Coll Cardiol. 2004;44:1393-9.

Intervention (Management)

- Based on risk stratification, patient was considered as very high risk for CIN (projected risk of 57.3%) with the risk expected to increase even further if contrast media (CM) was given in excess.
- Furosemide was held and patient was given normal saline at 1.5 mL/kg for 12 hours.
- Repeat serum creatinine was 1.4 mg/dL (reflected her baseline from 4 months ago).
- At that time, an angiogram was performed which revealed an in-stent restenosis of left circumflex that was treated with a drug-eluting stent.
- A 90% lesion was also noted in the right coronary artery (RCA), however the lesion was not treated so as to minimize the amount of CM.
- Postprocedure fluids were continued at the same rate for an additional 6 hours.

Outcomes

- Patient was monitored in the hospital for an additional 3 days.
- Her volume status was optimized, and prior to discharge, she was restarted on furosemide albeit at a lower dose.
- During her hospital stay, metabolic panel revealed no creatinine elevation, and on discharge, her creatinine was 1.2 mg/dL.

Learning Points

- Patients should always get risk stratified for CIN prior to angiography.
- If possible, the volume status of the patient should be optimized prior to angiography.
- Hydration with normal saline should be given to all patients before and after the procedure.
- The amount of CM should be minimized, and if further nonurgent intervention is needed, a subsequent staged procedure should be scheduled.

CASE 3: POST-PROCEDURE BLEEDING AND VASCULAR COMPLICATION

Ziad Sergie, Jennifer Yu, Roxana Mehran, Gregg W Stone

History

- A 63-year-old female presented to the hospital with recent onset of chest discomfort with minimal exertion, relieved with rest.
- *Past medical history:* Obesity, hypertension, hypercholesterolemia, diverticulosis (with recent gastrointestinal bleeding), gastroesophageal reflux.
- *Medications:* Amlodipine 5 mg daily, metoprolol tartrate 25 mg twice daily, atorvastatin 10 mg daily, lansoprazole 15 mg daily.
- *Allergies:* None.
- *Family history:* Negative for coronary artery disease.
- Physical examination was unremarkable.

Basic Investigations

- *CBC:* White count $8.6 \times 10^3/\text{uL}$, hemoglobin 12.3 g/dL, hematocrit 39.2%, platelet count $225 \times 10^3/\text{uL}$.
- *Chemistries:* Sodium 138 mEq/L, potassium 4.4 mEq/L, blood urea nitrogen 13 mg/dL, creatinine 0.8 mg/dL.
- *Coagulation parameters:* APTT 28 seconds, INR 0.9.
- *Nuclear stress test:* Modified Bruce protocol to 78% maximal heart rate (5 minutes), terminated because of chest pain (no ECG changes). Myocardial perfusion imaging revealed extensive apicolateral, inferolateral, and posterolateral ischemia.

Cardiac Catheterization

- *Access:* Right femoral artery
- *Anatomy:* Right dominant, two-vessel coronary artery disease (distal RCA subtotal occlusion with a large vessel distally, RPL 60–70%; OM1 subtotal occlusion with a moderate size vessel distally)
- *Ventriculogram:* Ejection fraction 50%, moderate hypokinesis of diaphragmatic and posterobasal segments
- *Pharmacology:* Aspirin 325 mg, clopidogrel 600 mg, bivalirudin IV (0.75 mg/kg × 1, then 1.75 mg/kg/h).
- *Percutaneous coronary intervention (PCI):* Bare metal stent 3.0 mm diameter, 23 mm long implanted in the distal RCA.
- Sheath was removed 2 hours post-PCI, and access site was managed with manual and mechanical compression.

Outcomes

- The patient was admitted overnight, and was noted to have a large right groin hematoma the following morning.
- Lower extremity ultrasound: right inguinal hematoma 7 × 5 cm, and 3.2 × 2 cm pseudoaneurysm of the distal common femoral artery (at the origin of the superficial femoral artery).
- She received ultrasound guided thrombin injection, with resolution of the pseudoaneurysm.
- Her nadir hemoglobin was 8.4 g/dL (nadir hematocrit 26.6%).
- She received two units of packed red blood cells, and her hemoglobin stabilized at 10.2 g/dL prior to discharge (hematocrit 31.1%).
- There was no acute cardiac event or recurrent bleeding at 1-year follow-up.

Learning Points

- Her bleeding complication can be classified as BARC type 3a (overt bleeding plus a hemoglobin drop 3 to ≤ 5 g/dL).
- Her estimated bleeding risk based on her clinical presentation was in the intermediate range (NCDR score of 55, >2% but ≤6.5% rate of postPCI major bleeding).
- Access site bleeding might have been averted with the use of radial artery access or a vascular closure device.
- A bare metal stent was implanted because of her high risk of recurrent gastrointestinal bleeding (to minimize the duration of dual antiplatelet therapy).
- The access site complication was managed without discontinuation of dual antiplatelet therapy.
- Whether blood transfusion was warranted in this case is uncertain.

CASE 4: BARC TYPE 2 BLEEDING IN A HIGH RISK PATIENT

Ziad Sergie, Jennifer Yu, Roxana Mehran, Gregg W Stone

History

- A 52-year-old female presented with sudden onset chest pressure and dyspnea at rest.
- *Past medical history:* Coronary artery disease (PCI to distal LCx 8 months ago), hypertension, hypercholesterolemia, smoking, end-stage renal disease on hemodialysis for 5 years (via a right AV fistula), medication nonadherence
- *Cardiac medications:* Aspirin 81 mg daily, clopidogrel 75 mg daily, labetalol 400 mg twice daily, clonidine 0.1 mg twice daily, nifedipine ER 90 mg daily, simvastatin 40 mg daily.
- *Allergies:* None.
- *Family history:* Her father had a MI at age 54.
- In the emergency room, her blood pressure was 190/115 mm Hg, heart rate 112 beats per minute, respiratory rate 22 breaths per minute, and oxygen saturation 92% on 3L nasal oxygen.
- *Physical examination:* Elevated jugular venous pressure, tachycardia with a S3 gallop, bibasilar rales, lower extremity edema.

Basic Investigations

- *CBC*: White count $9.4 \times 10^3/\mu\text{L}$, hemoglobin 9.8 g/dL, hematocrit 29.6%, platelet count $197 \times 10^3/\mu\text{L}$.
- *Chemistries*: Sodium 136 mEq/L, potassium 4.8 mEq/L, blood urea nitrogen 52 mg/dL, creatinine 8.8 mg/dL.
- *Coagulation parameters*: APTT 33 seconds, INR 1.1
- *Troponins*: 0.1 ng/mL (negative $\times 3$).
- *ECG*: Sinus tachycardia, left ventricular hypertrophy, ST depressions in the inferior leads, prolonged QTc (487 ms).
- *Chest X-ray*: Cardiomegaly, small area of atelectasis in the right base, no pleural effusions.
- *Echocardiogram 3 months prior*: LVEF 42% (diffuse), severe LVH, mild aortic stenosis

Cardiac Catheterization

- After treatment of hypertension with IV nitroglycerin infusion, coronary angiography was performed because of persistent symptoms.
- *Access*: Right femoral artery.
- *Anatomy*: Left dominant, two-vessel coronary artery disease; 80–90% in-stent restenosis at former DES site of distal LCx, large vessel distally; 80–90% stenosis in the proximal RCA (small nondominant vessel) not treated.
- *Pharmacology*: Aspirin 325 mg, clopidogrel 300 mg, bivalirudin [$0.75 \text{ mg/kg} \times 1$, then 0.25 mg/kg/h (adjusted for dialysis)].
- *PCI*: Cutting balloon 3.5 mm diameter, 6 mm long inflated at 12 ATM for 10 seconds at distal LCx ISR with good result.
- *Hemostasis*: Vascular closure device used.

Outcomes

- The following day, she received a hemodialysis session that was terminated early because of hypotension.
- Access site bleeding occurred after hemodialysis requiring mechanical compression.
- The bleeding ultimately responded to the topical application of thrombin powder.
- Following hemostasis, there was no evidence of hematoma or pseudoaneurysm at the access site.
- Her nadir hemoglobin was 8.3 g/dL (nadir hematocrit 24.8%).
- She subsequently tolerated hemodialysis and was discharged home.

Learning Points

- This bleeding event can be classified as BARC type 2 (overt, actionable sign of hemorrhage).
- The estimated bleeding risk based on her clinical presentation was high (NCDR score of 80, or $>6.5\%$ rate of post-PCI major bleeding).
- Bleeding avoidance strategies were employed in this case including the appropriate renal dosing of bivalirudin and successful deployment of a vascular closure device.
- Her right radial artery could not be catheterized because of the pre-existing right AV fistula; however left radial access might have been an option.
- Note that blood transfusion was not required.