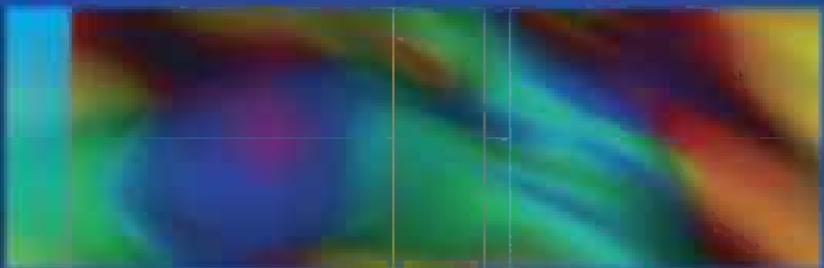


Annette Vegas



**Perioperative  
Two-Dimensional  
Transesophageal  
Echocardiography**  
A Practical Handbook

 Springer

# Perioperative Two-Dimensional Transesophageal Echocardiography



Annette Vegas

# Perioperative Two-Dimensional Transesophageal Echocardiography

A Practical Handbook



Springer

Annette Vegas, MD, FRCPC, FASE  
Associate Professor of Anesthesiology  
Director of Perioperative TEE  
Department of Anesthesia  
Toronto General Hospital  
University of Toronto  
M5G 2C4 Toronto  
Canada

#### Illustration Credits

Frances Yeung: pp. 200, 205.

Gian-Marco Busato: pp. 2, 3, 4, 6-26, 28, 43, 52-55, 59, 61, 63, 68, 70, 71, 74, 78, 79, 81, 88, 92-95, 99, 100, 101, 108, 109, 113, 121, 122, 126, 138, 140, 142, 147, 148, 149, 155, 157, 158, 159, 161, 164-176, 193, 195, 197, 200, 206, 207, 211, 212, 220, 221.

Maureen Wood: pp. 81, 90, 91, 132, 133, 135, 143.

Michael Corrin: p. 68.

Willa Bradshaw: pp. 27, 28, 47, 48, 87, 132, 153, 183, 185.

#### Photo Credits

Dr. R.J. Cusimano: pp. 186, 188-190.

Dr. T. David: pp. 187, 194.

Dr. Marc de Perrot: p. 191.

Dr. C. Feindel: p. 144.

ISBN 978-1-4419-9951-1

e-ISBN 978-1-4419-9952-8

DOI 10.1007/978-1-4419-9952-8

Springer New York Dordrecht Heidelberg London

Library of Congress Control Number: 2011928399

© Springer Science+Business Media, LLC 2012

All rights reserved. This work may not be translated or copied in whole or in part without the written permission of the publisher (Springer Science+Business Media, LLC, 233 Spring Street, New York, NY 10013, USA), except for brief excerpts in connection with reviews or scholarly analysis. Use in connection with any form of information storage and retrieval, electronic adaptation, computer software, or by similar or dissimilar methodology now known or hereafter developed is forbidden.

The use in this publication of trade names, trademarks, service marks, and similar terms, even if they are not identified as such, is not to be taken as an expression of opinion as to whether or not they are subject to proprietary rights.

While the advice and information in this book are believed to be true and accurate at the date of going to press, neither the authors nor the editors nor the publisher can accept any legal responsibility for any errors or omissions that may be made. The publisher makes no warranty, express or implied, with respect to the material contained herein.

Printed on acid-free paper

Springer is part of Springer Science+Business Media ([www.springer.com](http://www.springer.com))

# Dedication

To my parents, Patrick and Lena, and my brother Derek for their love and support throughout my life.

To colleagues at Toronto General Hospital, in particular Dr Christopher Feindel, cardiac surgeon, and Dr Patricia McNama, anesthesiologist, who have been exemplars of professionalism during my career.



# Preface

The role of transesophageal echocardiography (TEE) has expanded to become common place during cardiac surgery and in the ICU. Anesthesiologists trained in TEE are increasingly providing this service in both venues. The skills and expertise of the echocardiographer are constantly evolving to provide timely and accurate information. The challenge for the echocardiographer is to integrate many current TEE guidelines into everyday practice. There is a need to have reference material readily at hand to confirm echocardiographic findings. This handbook is created to fulfill the need for an illustrative synopsis of common cardiac pathology encountered in cardiac surgery patients. It is designed to provide a compact portable reference for using TEE to recognize cardiac pathology in the perioperative period. It will appeal to anesthesiologists, cardiac surgeons and cardiologists with a range of experience from novice to expert echocardiographers.

This handbook is a compilation of echocardiography information and TEE images from perioperative TEE studies performed at Toronto General Hospital (TGH), Toronto, Ontario, Canada. As with all written texts it does not do justice to the cardiac activity seen in live TEE. The reader is referred to other sources for video recordings of TEE. The TEE website, <http://pie.med.utoronto.ca/TEE/> developed by the Perioperative Interactive Education (PIE) group at Toronto General Hospital is a rich online resource for TEE educational material. Readers who prefer a more traditional source can view a reference textbook such as Multimedia Transesophageal Echocardiography 2<sup>nd</sup> Edition (2010) published by Informa Healthcare and edited by Drs Andre Denault, Pierre Couture, Annette Vegas, Jean Buithieu and Jean Claude Tardif.

Learning and practicing echocardiography is a career-long process. In the words of Galileo Galilei, "You cannot teach a man anything; you can only help him to find it for himself." I hope this handbook will help you along your journey.

Dr. Annette Vegas, MD, FRCPC, FASE  
January 2011



# Acknowledgments

To members of the current TGH Anesthesiology Perioperative TEE group: Drs. L. Bahrey, G. Djaiani, J. Heggie, M. Jariani, J. Karski, R. Katznelson, P. McNama, M. Meineri, P. Murphy, P. Slinger, A. Van Rensburg and M. Wasowicz who perform, train and educate others about TEE.

To my colleagues from the Division of Cardiac Surgery, under the leadership of Dr. Tirone David, who attract a varied practice that keeps TGH cardiac anesthesiologists challenged to provide exemplary patient care.

To Ms. Jo Carroll, Manager, Anesthesia Research, Department of Anesthesia & Pain Management, Toronto General Hospital for being a pillar of moral support throughout the years.

To members of the TGH cardiology echocardiography lab, under the direction of Dr. Anna Woo and former directors Dr. Sam Siu and Dr. Harry Rakowski who generously share their knowledge with the perioperative TEE group at TGH.

To Lizette Bicular, UHN PMCC Echocardiography Laboratory Manager, a skilled sonographer with the patience of a saint.

To Dr. Doris Basic, staff cardiologist from the University of Calgary, for her review and insightful comments in preparing previous versions of this manuscript.

To medical student Mr. Gian-Marco Busato MSc, for his extraordinary artistic talent he used to draw the illustrations for this handbook.

Finally to Ms. Willa Bradshaw BSc MScBMC, medical illustrator, who precisely assembled all the detailed figures.



# Contents

<b>1 Normal TEE Views.....</b>	<b>1</b>
<b>2 Doppler and Hemodynamics.....</b>	<b>31</b>
<b>3 Ventricles .....</b>	<b>51</b>
<b>4 Native Valves .....</b>	<b>77</b>
<b>5 Prosthetic Valves, Transcatheter Valves and Valve Repairs .....</b>	<b>117</b>
<b>6 Aorta.....</b>	<b>137</b>
<b>7 Congenital Heart Disease .....</b>	<b>151</b>
<b>8 Variants, Foreign Material, Masses and Endocarditis.....</b>	<b>179</b>
<b>9 Ventricular Assist Devices and Heart Transplantation .....</b>	<b>199</b>
<b>10 Hypertrophic Obstructive Cardiomyopathy and Diastolic Dysfunction .....</b>	<b>209</b>
<b>11 Pericardium.....</b>	<b>219</b>
<b>Index.....</b>	<b>229</b>



# Abbreviations

A	Anterior
AI	Aortic insufficiency
AL	Anterolateral
AMVL	Anterior mitral valve leaflet
AS	Aortic stenosis
ASD	Atrial septal defect
ASE	American Society of Echocardiography
AV	Aortic valve
AVA	Aortic valve area
AVSD	Atrioventricular septal defect
BAV	Bicuspid aortic valve
BPM	Beats per minute
C	Chamber
CAD	Coronary artery disease
CE	Carpentier-Edwards
CO	Cardiac output
CPB	Cardiopulmonary bypass
CS	Coronary sinus
CSA	Cross sectional area
CVP	Central venous pressure
CW	Continuous wave
Cx	Circumflex artery
DS	Deceleration slope
DT	Deceleration time
DVI	Dimensionless valve index
ED	End diastole
EDA	End diastolic area
EDD	End diastolic diameter
EDP	End diastolic pressure
EDV	End diastolic volume
EF	Ejection fraction
ERO	Effective regurgitant orifice
ES	End systole
ESA	End systolic area
ESD	End systolic diameter
ESV	End systolic volume
FAC	Fractional area change
FS	Fractional shortening
GE	Gastroesophageal

HBP	High blood pressure
HOCM	Hypertrophic obstructive cardiomyopathy
HR	Heart rate
HV	Hepatic vein
I	Inferior
IABP	Intra-aortic balloon pump
IAS	Inter-atrial septum
IHSS	Idiopathic hypertrophic subaortic stenosis
IPPV	Intermittent positive pressure ventilation
IVC	Inferior vena cava
IVRT	Isovolumetric relaxation time
IVS	Interventricular septum
JA	Jet area
JH	Jet height
L	Left or lateral or length
LA	Left atrium
LAA	Left atrial appendage
LAD	Left anterior descending
LAP	Left atrial pressure
LAX	Long axis
LCA	Left coronary artery
LCC	Left coronary cusp
LCCA	Left common carotid artery
LLPV	Left lower pulmonary vein
LUPV	Left upper pulmonary vein
LV	Left ventricle
LVAD	Left ventricular assist device
LVH	Left ventricular hypertrophy
LVID	Left ventricle internal diameter
LVOT	Left ventricular outflow tract
MAC	Mitral annular calcification
MI	Myocardial infarction
MR	Mitral regurgitation
MS	Mitral stenosis
MVA	Mitral valve area
N	Non
NSR	Normal sinus rhythm
P	Pressure or posterior
PA	Pulmonary artery
PAP	Pulmonary artery pressure
PAPVD	Partial anomalous pulmonary venous drainage
PASP	Pulmonary artery systolic pressure
PDA	Patent ductus arteriosus
PFO	Patent foramen ovale
PHT	Pressure half-time
PI	Pulmonic insufficiency

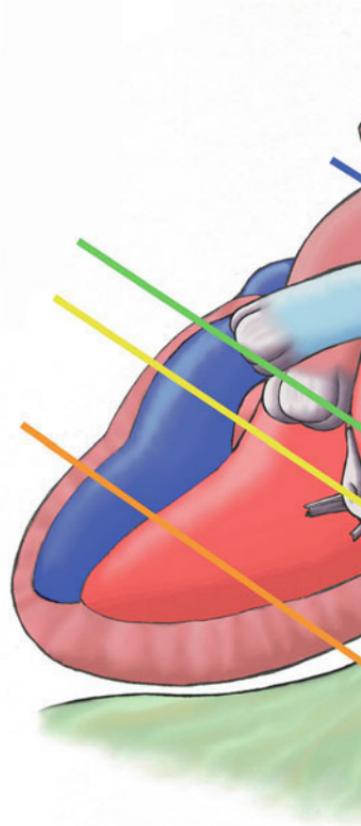
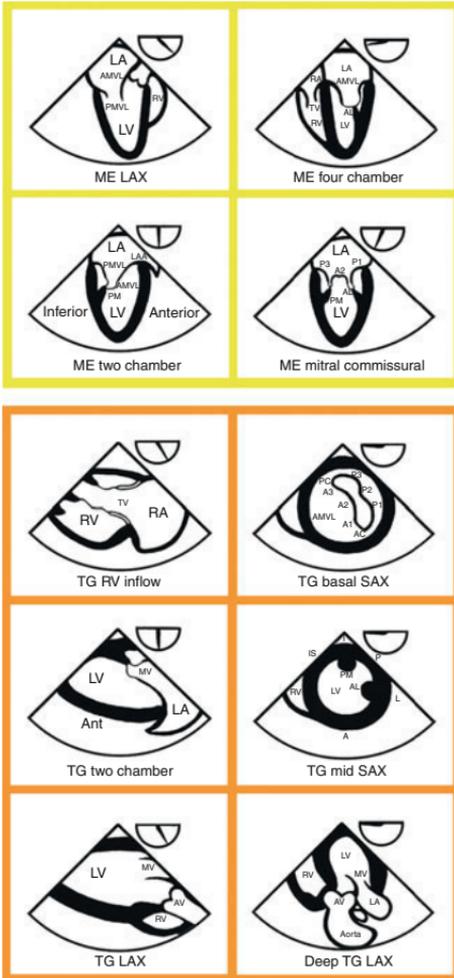
PISA	Proximal isovelocity surface area
PM	Papillary muscles or posteromedial
PMVL	Posterior mitral valve leaflet
Pr	Prosthetic
PS	Pulmonic stenosis
PV	Pulmonic valve
PW	Pulsed wave
R	Right
RA	Right atrium
RAP	Right atrial pressure
RCA	Right coronary artery
RCC	Right coronary cusp
RegV	Regurgitant volume
RF	Regurgitant fraction
RLPV	Right lower pulmonary vein
RUPV	Right upper pulmonary vein
RV	Right ventricle
RVH	Right ventricular hypertrophy
RVOT	Right ventricular outflow tract
RVSP	Right ventricular systolic pressure
SAM	Systolic anterior motion
SAX	Short axis
SC	Saline contrast
SCA	Society of Cardiovascular Anesthesiology
SLCL	Septal leaflet contact length
SLE	Systemic lupus erythematosus
SOVA	Sinus of Valsalva aneurysm
SPV	Stentless porcine valve
STJ	Sinotubular junction
SV	Stroke volume
SVA	Systemic venous atrium
SVC	Superior vena cava
SWMA	Segmental wall motion abnormality
TEE	Transesophageal echocardiography
TG	Transgastric
TGA	Transposition of the great arteries
TOF	Tetralogy of Fallot
TR	Tricuspid regurgitation
TS	Tricuspid stenosis
TTE	Transthoracic echocardiography
TV	Tricuspid valve
UE	Upper esophageal
VSD	Ventricular septal defect
VTI	Velocity time integral
W	Width
WPW	Wolf Parkinson White



# Normal TEE Views

Overview 20 Standard TEE Views.....	2,3
TEE Planes and Display.....	4
Standard TEE Views Guide.....	5
Mid-esophageal Four Chamber (ME 4C).....	6
Mid-esophageal Mitral Commissural (ME MC).....	7
Mid-esophageal Two Chamber (ME 2C).....	8
Mid-esophageal Long-Axis (ME LAX).....	9
Mid-esophageal Aortic Valve Long-Axis (ME AV LAX).....	10
Mid-esophageal Aortic Valve Short-Axis (ME AV SAX).....	11
Mid-esophageal Right Ventricular Outflow (ME RVOT).....	12
Mid-esophageal Bicaval.....	13
Transgastric Basal Short-Axis (TG Basal SAX).....	14
Transgastric Mid Short-Axis (TG Mid SAX).....	15
Transgastric Two Chamber (TG 2C).....	16
Transgastric Long-Axis (TG LAX).....	17
Transgastric Deep Long-Axis (TG Deep LAX).....	18
Transgastric Right Ventricular Inflow (TG RV Inflow).....	19
Mid-esophageal Descending Aortic Short-Axis (SAX).....	20
Mid-esophageal Descending Aortic Long-Axis (LAX).....	21
Upper-esophageal Aortic Arch Long-Axis (LAX).....	22
Upper-esophageal Aortic Arch Short-Axis (SAX).....	23
Mid-esophageal Ascending Aortic Short-Axis (SAX).....	24
Mid-esophageal Ascending Aortic Long-Axis (LAX).....	25
Mid-esophageal Five Chamber (ME 5C).....	26
Transgastric Inferior Vena Cava (TG IVC).....	27
Mid-esophageal Left Atrial Appendage (ME LAA).....	28
Transgastric Superior and Inferior Vena Cava.....	28
Mid-esophageal Coronary Sinus.....	29
Mid-esophageal Tricuspid Valve.....	29

## Overview 20 Standard TEE Views



The 20 basic TEE views as described by the SCA and ASE are diagrammed here. Conveniently, the views are grouped together by the structures being interrogated:

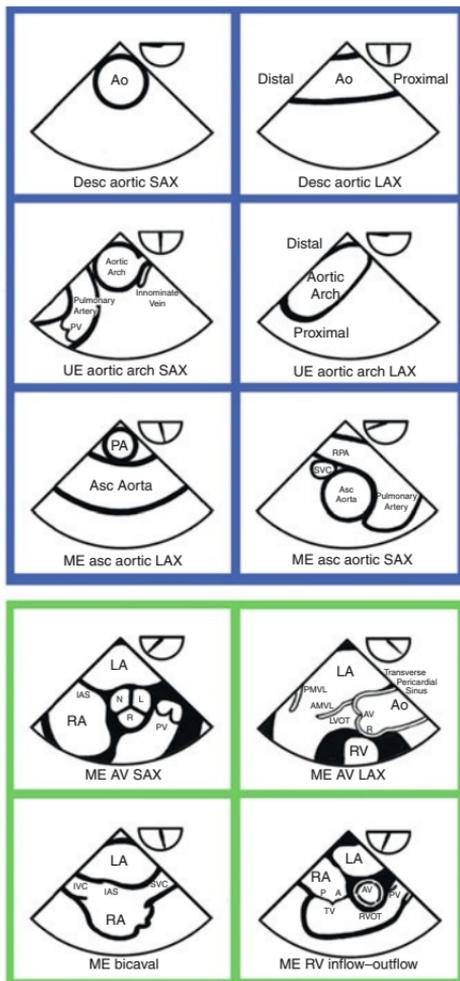
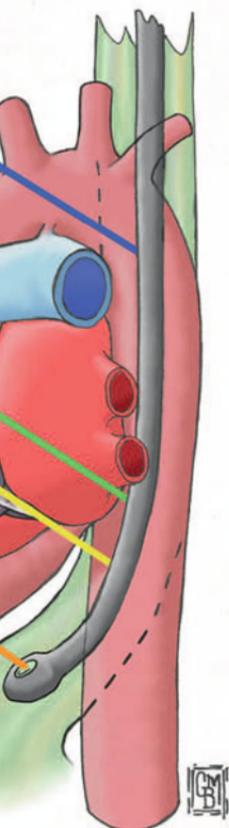
**Yellow:** Mid-esophageal (ME) views that image the LV and MV

**Orange:** Transgastric (TG) views that image the LV, RV, and AV for spectral Doppler alignment

**Blue:** ME and UE views that image different regions of the aorta

**Green:** ME views that image the AV, RVOT, and bicaval

## Overview 20 Standard TEE Views



### Sources

- **Shanewise JS, Cheung AT, Aronson S, et al.** ASE/SCA Guidelines for performing a comprehensive intraoperative multiplane transesophageal echocardiography examination. *Anesth Analg* 1999; 89:870-84.
- **Flachskampf FA, Decoodt P, Fraser AG, et al.** Guideline from the Working Group: Recommendations for Performing Transesophageal Echocardiography. *Eur J Echocardiograph* 2001; 2:8-21.

# TEE Planes and Display

## TEE Probe Manipulation

Probe movements (entire probe moves):

1. Advance or withdraw
2. Turn right or left

Knob movements (only probe tip moves):

3. Flex right or left
4. Anteflex or retroflex

Transducer movements (probe stays still):

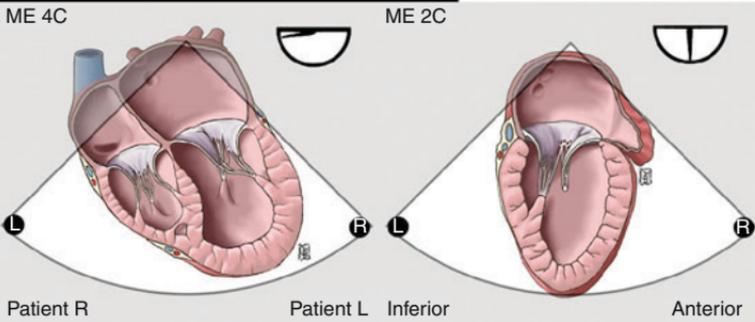
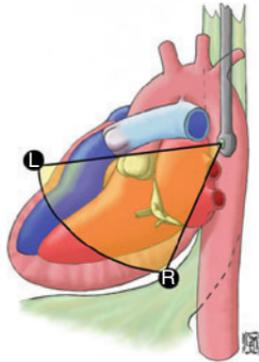
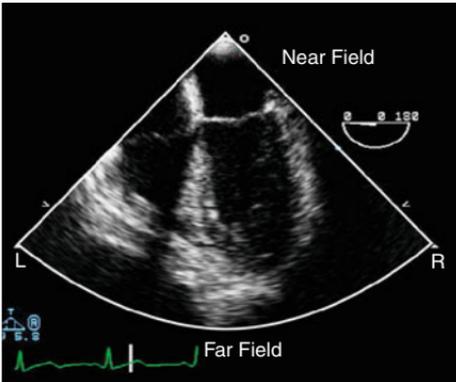
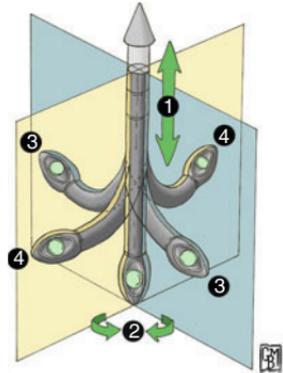
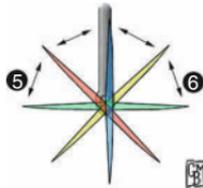
5. Rotate angle forward ( $0^{\circ}$ – $180^{\circ}$ )
6. Rotate angle back ( $180^{\circ}$ – $0^{\circ}$ )

## Transducer Planes

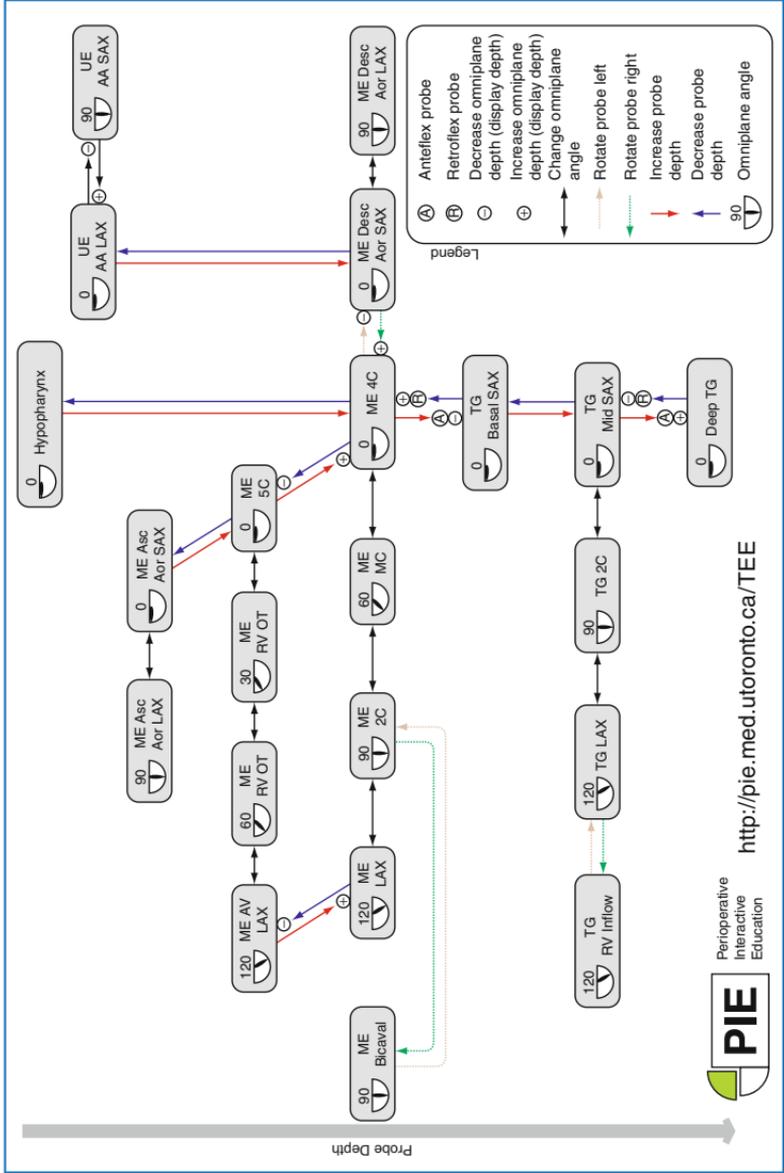
- Transverse ( $0^{\circ}$ )
- Longitudinal ( $90^{\circ}$ )
- Omniplane ( $0^{\circ}$ – $180^{\circ}$ )

## Image Display

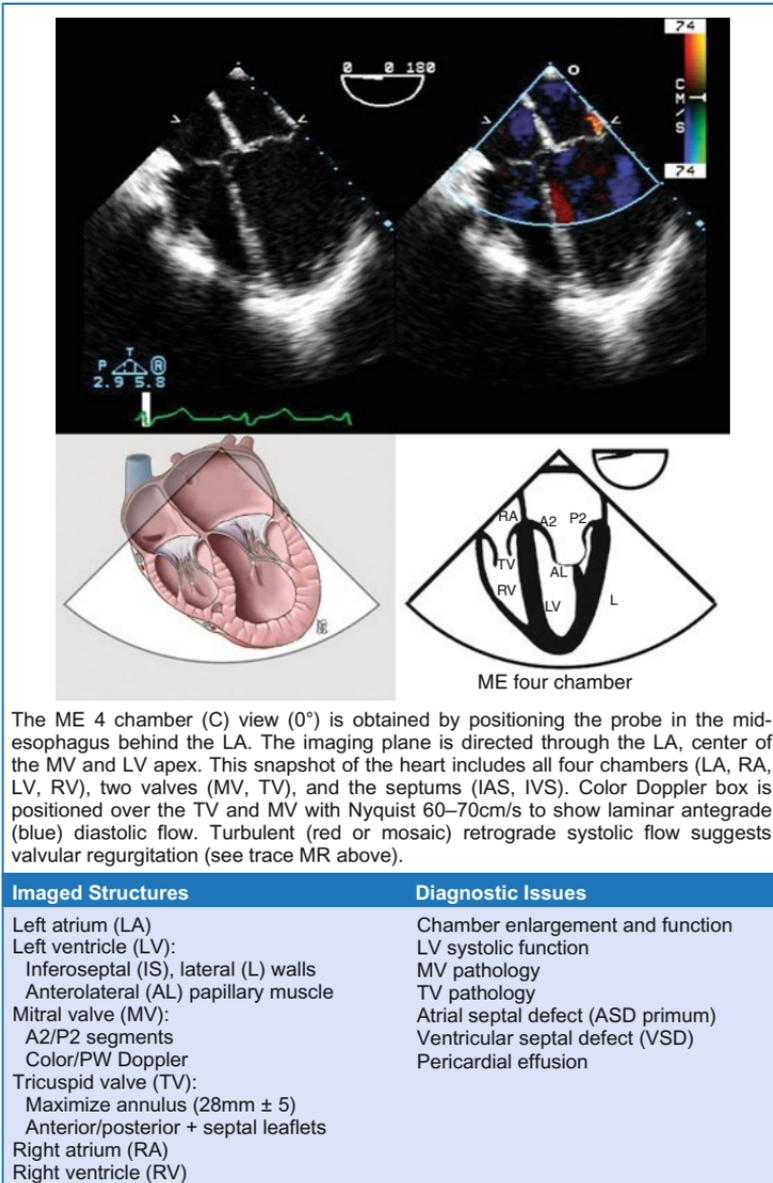
- Pie-shaped sector
- Display right (R), left (L)
- Near field (closest to probe)
- Far field (furthest from probe)



# Standard TEE Views Guide



## Mid-esophageal Four Chamber (ME 4C)



The ME 4 chamber (C) view (0°) is obtained by positioning the probe in the mid-esophagus behind the LA. The imaging plane is directed through the LA, center of the MV and LV apex. This snapshot of the heart includes all four chambers (LA, RA, LV, RV), two valves (MV, TV), and the septums (IAS, IVS). Color Doppler box is positioned over the TV and MV with Nyquist 60–70cm/s to show laminar antegrade (blue) diastolic flow. Turbulent (red or mosaic) retrograde systolic flow suggests valvular regurgitation (see trace MR above).

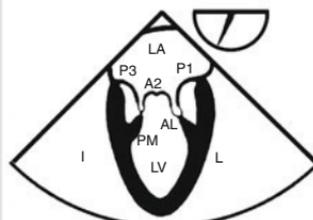
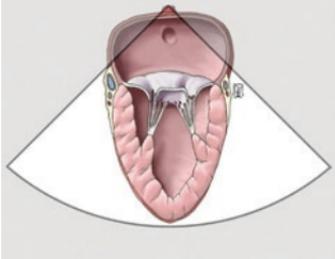
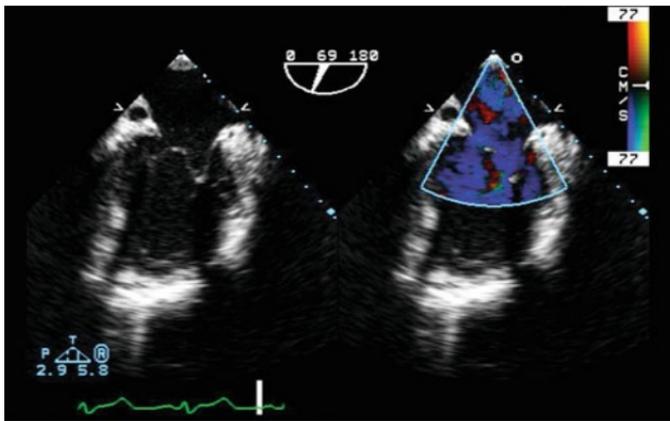
### Imaged Structures

Left atrium (LA)  
 Left ventricle (LV):  
   Inferoseptal (IS), lateral (L) walls  
   Anterolateral (AL) papillary muscle  
 Mitral valve (MV):  
   A2/P2 segments  
   Color/PW Doppler  
 Tricuspid valve (TV):  
   Maximize annulus (28mm ± 5)  
   Anterior/posterior + septal leaflets  
 Right atrium (RA)  
 Right ventricle (RV)

### Diagnostic Issues

Chamber enlargement and function  
 LV systolic function  
 MV pathology  
 TV pathology  
 Atrial septal defect (ASD primum)  
 Ventricular septal defect (VSD)  
 Pericardial effusion

## Mid-esophageal Mitral Commissural (ME MC)



ME mitral commissural

In the ME mitral commissural (MC) view, the crystals in the probe are now imaging at 45°–70° through the LA, center of the MV, and LV apex. The P3 scallop (left), P1 scallop (right), and A2 segment are in the middle forming the intermittently seen “trap door”. The probe is carefully manipulated to image both the posteromedial (PM) and anterolateral (AL) papillary muscles and LV apex. Color Doppler box is positioned over the MV with Nyquist 60–70cm/s to show laminar antegrade (blue) diastolic flow. Turbulent (red or mosaic) retrograde systolic flow suggests mitral regurgitation (MR). The high Nyquist of 77cm/s shown above will underestimate any MR.

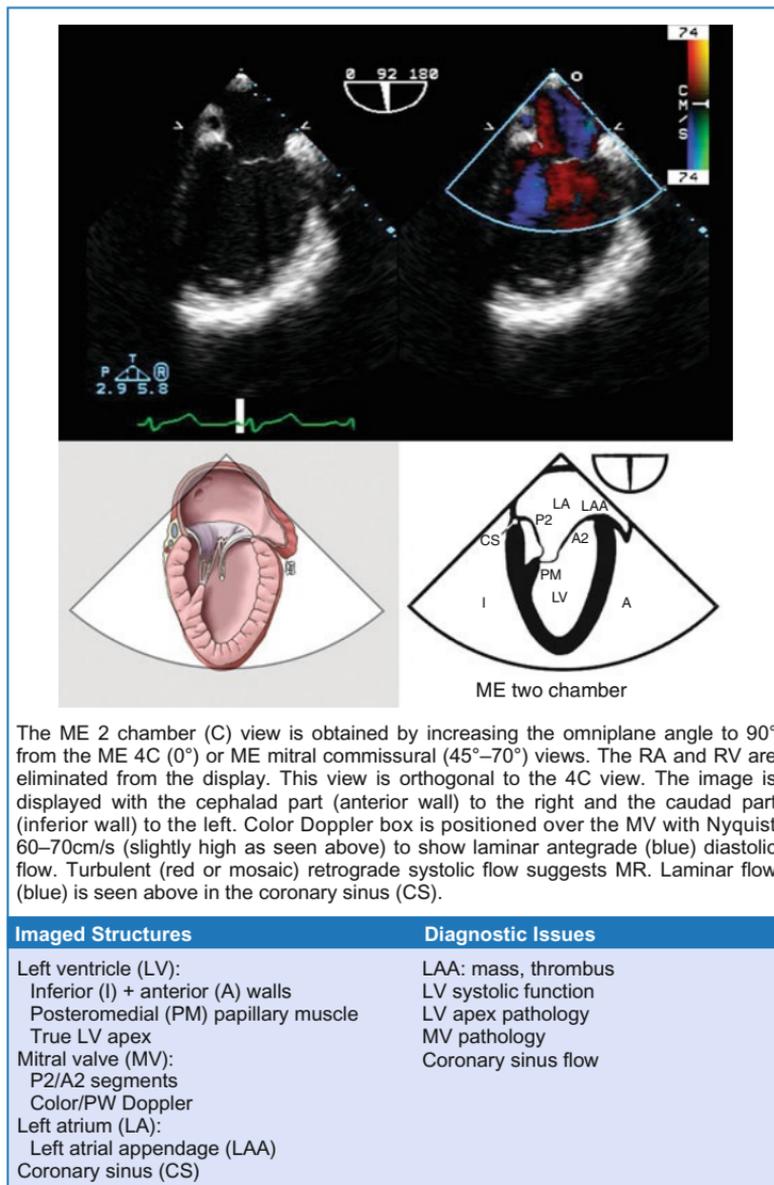
### Imaged Structures

Left ventricle (LV):  
 Inferior (I) + lateral (L) walls  
 Papillary muscles:  
     Posteromedial (PM)  
     Anterolateral (AL)  
 Mitral Valve (MV):  
     P3/A2/P1 segments  
     Color/PW Doppler  
 Left atrium (LA)  
 Coronary sinus  
 Circumflex artery

### Diagnostic Issues

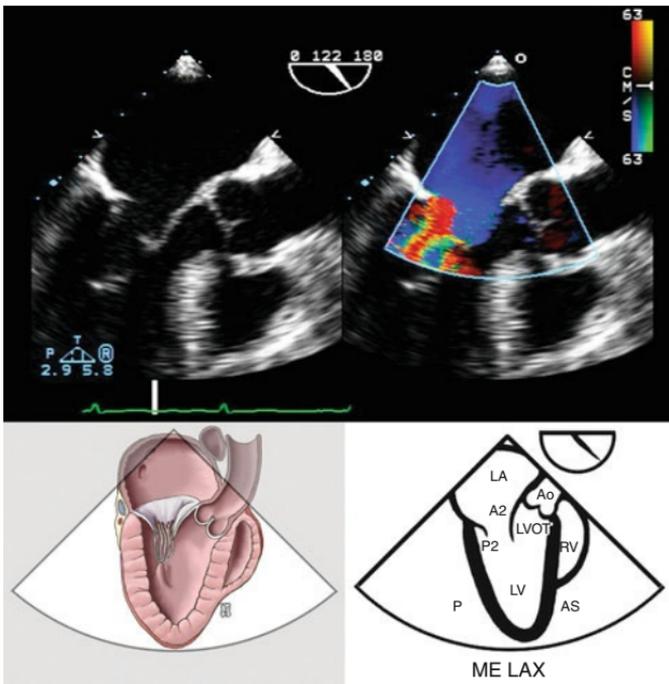
LA: mass, thrombus  
 LV systolic function  
 MV pathology  
 Coronary sinus flow

## Mid-esophageal Two Chamber (ME 2C)



The ME 2 chamber (C) view is obtained by increasing the omniplane angle to 90° from the ME 4C (0°) or ME mitral commissural (45°–70°) views. The RA and RV are eliminated from the display. This view is orthogonal to the 4C view. The image is displayed with the cephalad part (anterior wall) to the right and the caudad part (inferior wall) to the left. Color Doppler box is positioned over the MV with Nyquist 60–70cm/s (slightly high as seen above) to show laminar antegrade (blue) diastolic flow. Turbulent (red or mosaic) retrograde systolic flow suggests MR. Laminar flow (blue) is seen above in the coronary sinus (CS).

## Mid-esophageal Long-Axis (ME LAX)



The ME LAX view is obtained by increasing the omniplane angle to  $120^\circ$  from the ME 4C ( $0^\circ$ ) or ME MC ( $45^\circ$ – $70^\circ$ ) or ME 2C ( $90^\circ$ ) views. The more cephalad structures including the LVOT, AV, and proximal ascending aorta are lined up on the display right. The depth is adjusted to include the entire LV. Color Doppler box is positioned over the MV, LVOT, and AV with Nyquist 50–70cm/s to show laminar antegrade (blue) diastolic flow through the MV and systolic flow (red) through the LVOT and AV. Flow acceleration is seen above during diastole through this open MV.

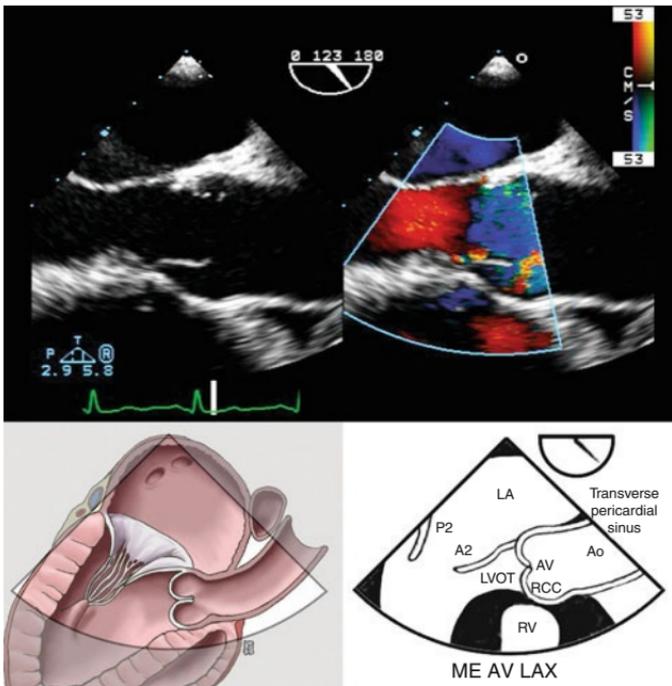
### Imaged Structures

Left atrium (LA)  
 Mitral valve (MV):  
   P2/A2 segments  
   Color/PW Doppler  
 Left ventricle (LV):  
   Posterior (P) + antero-septal (AS) walls  
   Not true LV apex  
 Interventricular septum (IVS)  
 Left ventricular outflow tract (LVOT)  
 Aortic valve (AV)  
 Aortic root and ascending aorta (Ao)

### Diagnostic Issues

MV pathology  
 LV systolic function  
 IVS pathology (VSD)  
 LVOT pathology  
 AV pathology  
 Aortic root pathology

## Mid-esophageal Aortic Valve Long-Axis (ME AV LAX)



The ME aortic valve LAX view ( $120^{\circ}$ – $130^{\circ}$ ) is obtained by decreasing the depth from the ME LAX view ( $120^{\circ}$ ). The LVOT, AV, and proximal ascending aorta are lined up on the display right, and the remainder of the MV and LV are eliminated from the image. The AV is seen in LAX with the anterior cusp always the right coronary cusp (RCC), the other is either the non or left coronary cusp. Color Doppler box is positioned over the AV with Nyquist 50–70cm/s to show laminar antegrade systolic flow. Though flow is continuous and unidirectional, in relation to the probe, it appears red through the LVOT and blue through the AV and ascending aorta.

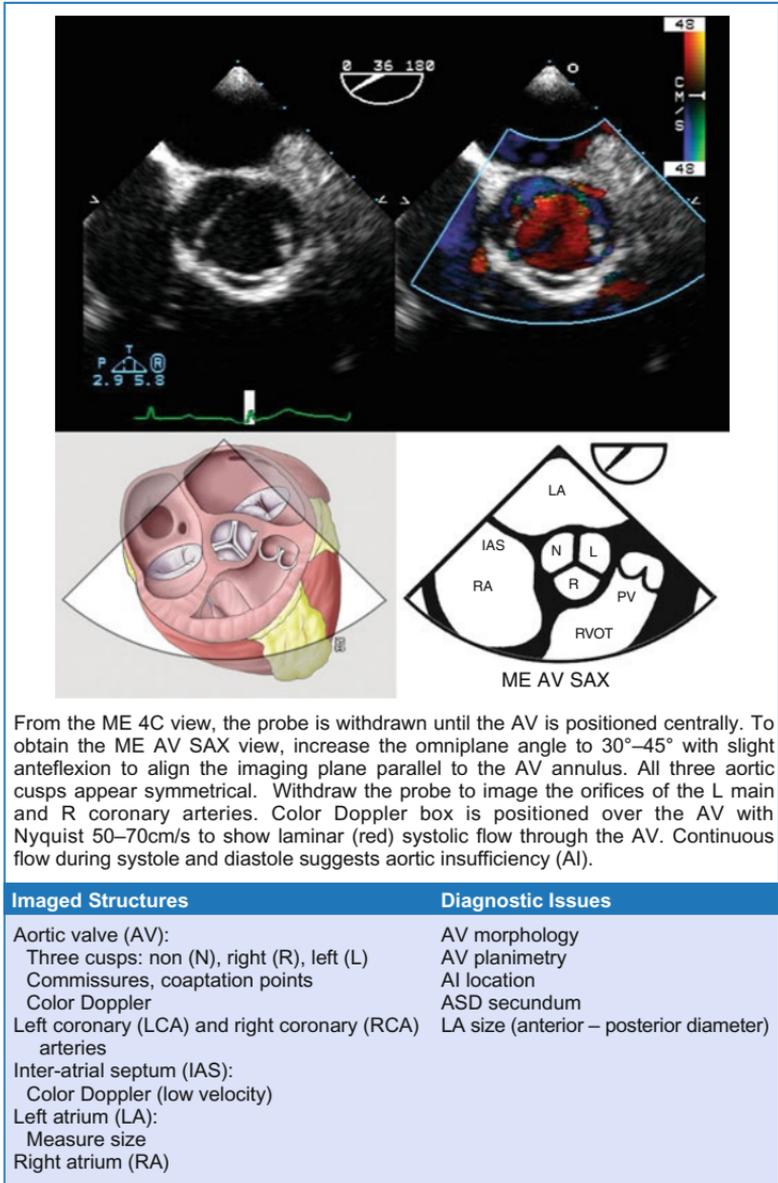
### Imaged Structures

Left ventricle (LV)  
 Left ventricular outflow tract (LVOT)  
 Aortic valve (AV):  
     Right cusp (RCC), left or non cusps  
     Color Doppler  
 Aortic root and ascending aorta (Ao)  
 Mitral valve (MV):  
     P2/A2 segments  
     Color/PW Doppler  
 Transverse pericardial sinus

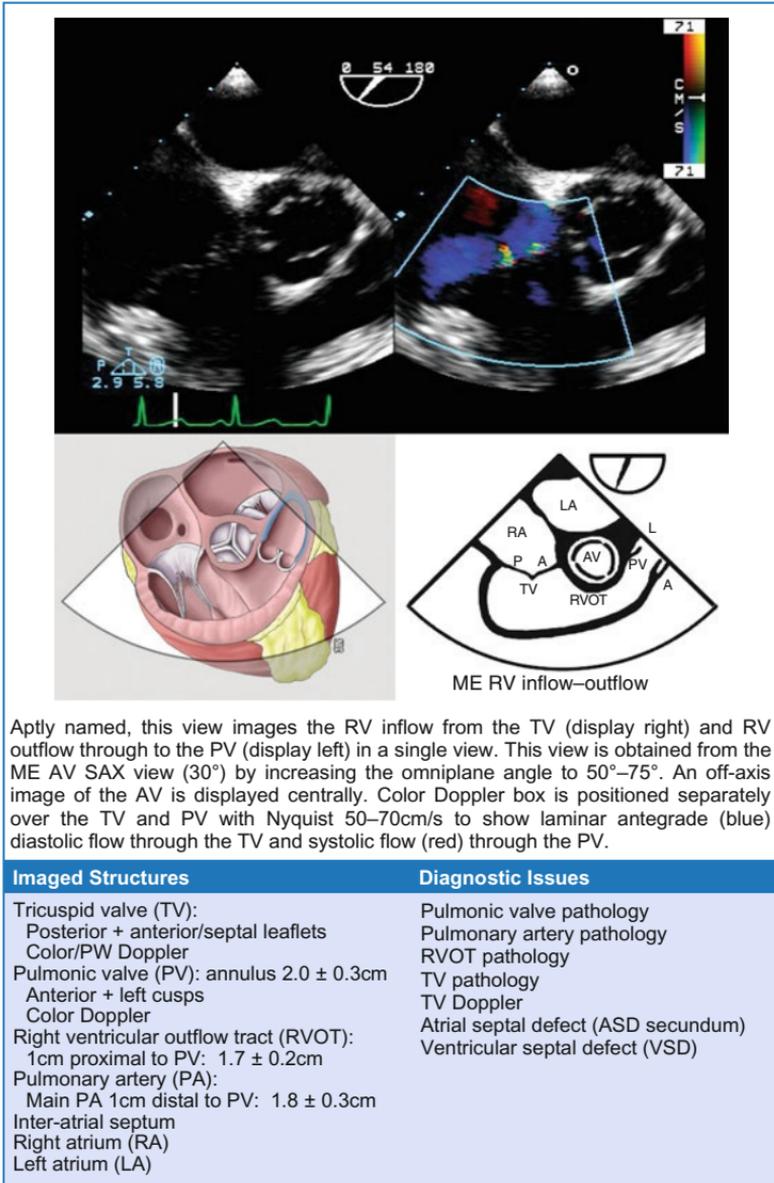
### Diagnostic Issues

AV pathology  
 Aortic root dimensions  
 Aortic root pathology  
 LVOT pathology  
 MV anterior leaflet  
 Ventricular septal defect (VSD)

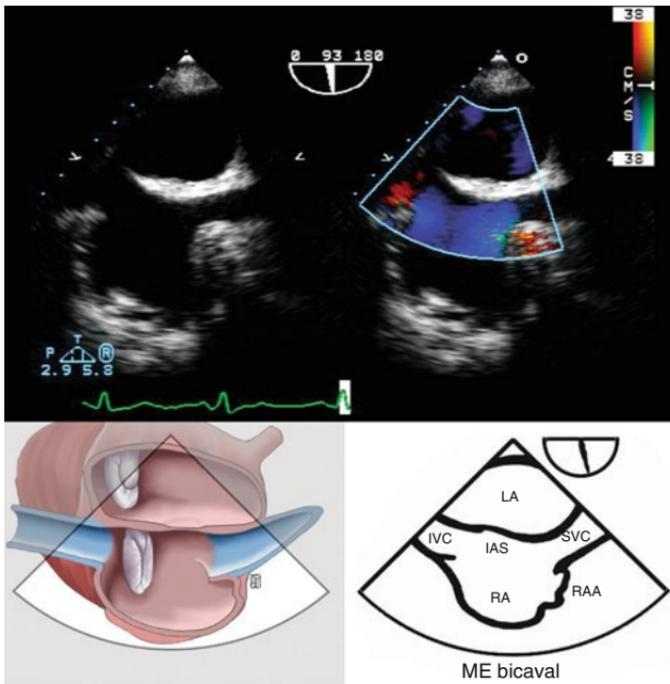
## Mid-esophageal Aortic Valve Short-Axis (ME AV SAX)



## Mid-esophageal Right Ventricular Outflow (ME RVOT)



## Mid-esophageal Bicaval



The ME bicaval view (90°) is obtained from the ME 2C view (90°) by turning the entire probe to the patient's right (towards the SVC and IVC). The transducer plane cuts through the LA, RA, and LAX of the IVC and SVC. The structures are displayed with the LA at the sector apex (closest to probe), RA in the far field, caudad IVC (left), and cephalad SVC (right). Color Doppler box is positioned over the IAS and proximal IVC and SVC with Nyquist 30–50cm/s. Laminar antegrade flow is present in the cava. Any flow across the IAS is abnormal suggesting an ASD or patent foramen ovale (PFO).

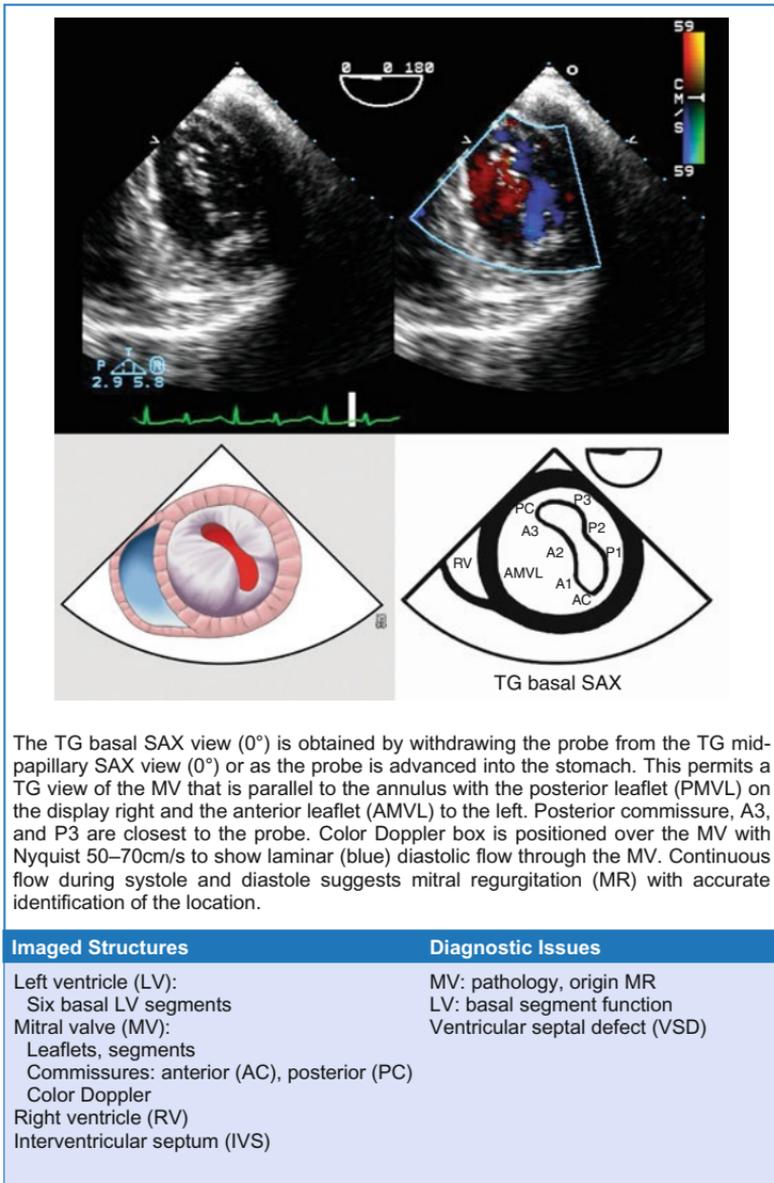
### Imaged Structures

Left atrium (LA)  
 Right atrium (RA):  
     Free wall, appendage (RAA)  
     Eustachian valve  
     Crista terminalis  
 Superior vena cava (SVC):  $1.4 \pm 0.2\text{cm}$   
 Inferior vena cava (IVC):  $1.6 \pm 0.2\text{cm}$   
 Inter-atrial septum (IAS):  
     Color Doppler (low velocity)

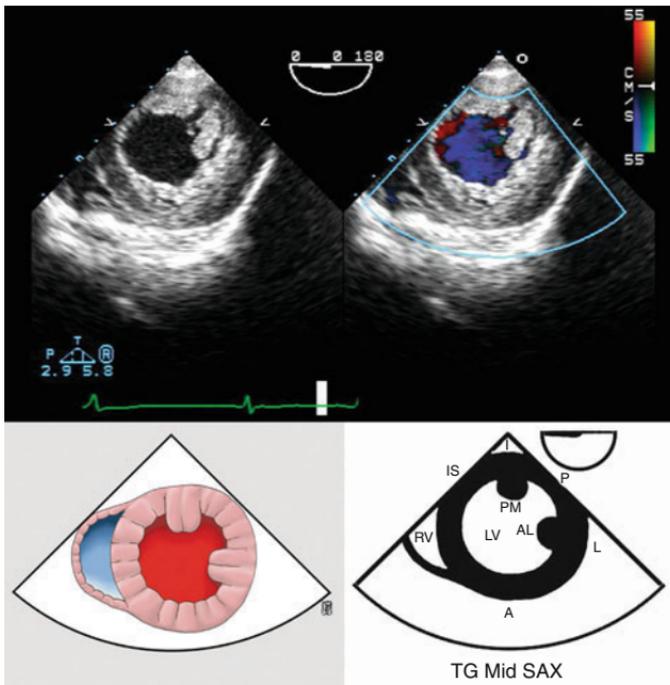
### Diagnostic Issues

Atrial septal defect (ASD)  
 Mass  
 SVC flow  
 IVC flow  
 Venous catheters  
 Pacemaker wires  
 Venous cannula position (SVC/IVC)

## Transgastric Basal Short-Axis (TG Basal SAX)



## Transgastric Mid Short-Axis (TG Mid SAX)



The TG views are obtained by advancing the TEE probe in a neutral position into the stomach and applying varying degrees of ante-flexion. In the TG mid papillary SAX view ( $0^\circ$ ), the LV is imaged in SAX with all six LV segments viewed at once. Manipulate the probe to center the LV cavity and slightly increase the transducer angle to obtain a symmetrical circular LV with both papillae present. Color Doppler box is positioned in the middle of the LV or over the IVS with Nyquist 50–70cm/s. Though not frequently used, color Doppler can be used to show flow across the IVS suggesting a ventricular septal defect (VSD).

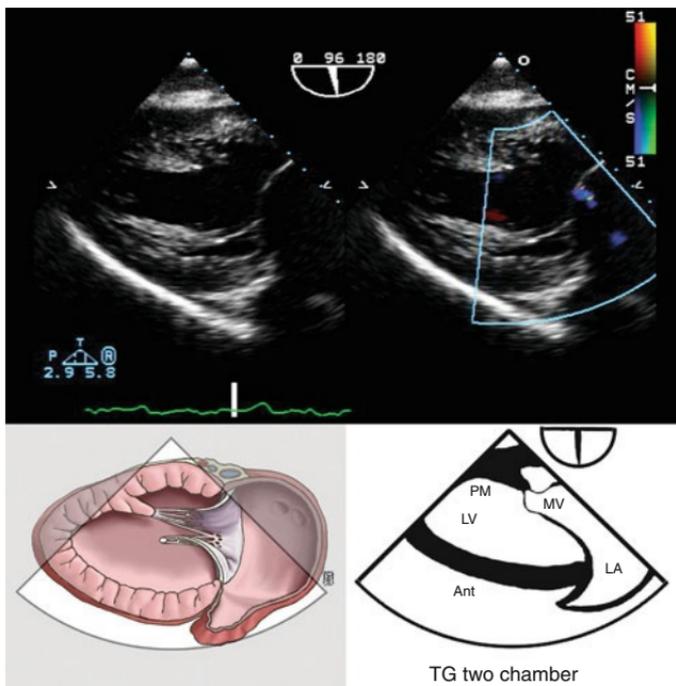
### Imaged Structures

Left ventricle (LV): opposing mid segments  
 Inferior (I) ↔ anterior (A)  
 Posterior (P) ↔ antero-septal (AS)  
 Lateral (L) ↔ infero-septal (IS)  
 Papillary muscles:  
 Anterolateral (AL)  
 Posteromedial (PM)  
 Right ventricle (RV)  
 Interventricular septum (IVS)

### Diagnostic Issues

LV cavity size  
 LV wall thickness  
 LV systolic function  
 Hemodynamic instability  
 IVS motion  
 Ventricular septal defect (VSD)

## Transgastric Two Chamber (TG 2C)



TG two chamber

The TG 2 chamber (C) view is obtained from the TG mid-papillary SAX view ( $0^\circ$ ) by increasing the transducer angle to  $75^\circ$ – $100^\circ$ . This images the LV in LAX and the subvalvular structures of the mitral valve. This view is similar to the ME 2 chamber view now turned  $90^\circ$  with the probe closest to the inferior wall of the LV (sector apex). Color Doppler box is positioned over the MV with Nyquist 50–70cm/s. Laminar diastolic flow occurs through the MV. Retrograde systolic flow suggests mitral regurgitation (MR).

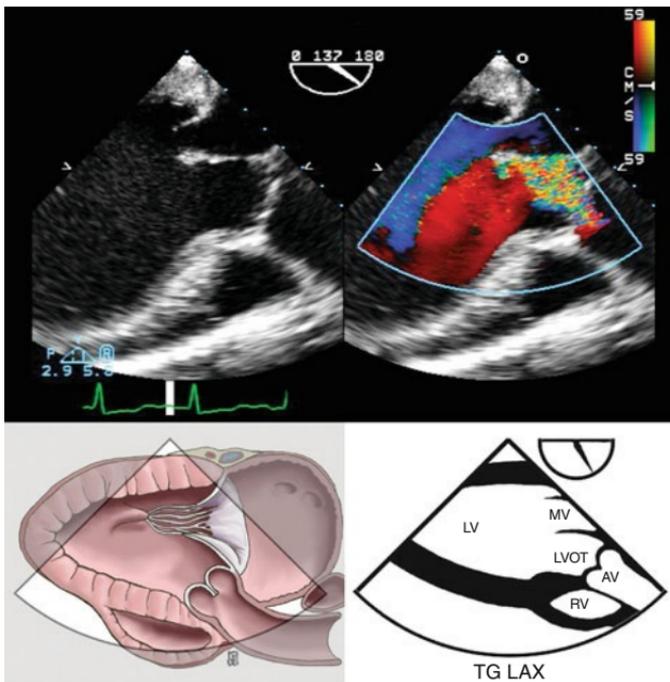
### Imaged Structures

Left ventricle (LV):  
 Apex  
 Anterior + inferior walls (basal + mid segments)  
 Posteromedial papillary muscle (PM)  
 Left atrium (LA):  
 Left atrial appendage (LAA)  
 Mitral valve (MV):  
 Leaflets (anterior and posterior)  
 Subvalvular apparatus  
 Color doppler

### Diagnostic Issues

LV systolic function  
 MV subvalvular apparatus  
 MV pathology

## Transgastric Long-Axis (TG LAX)



The TG LAX view is developed from the TG 2C view (90°) by increasing the transducer angle to 120°–140°. The LVOT and AV appear on the display right, depending on the depth settings. This view is similar to the ME AV LAX view and permits better spectral Doppler alignment. Color Doppler box is positioned over the MV, LVOT, and AV with Nyquist 50–70cm/s to show laminar antegrade systolic flow through the LVOT (red) and AV (blue). Turbulent diastolic flow through the AV suggests aortic insufficiency (AI) as shown above; systolic flow through the IVS represents a VSD.

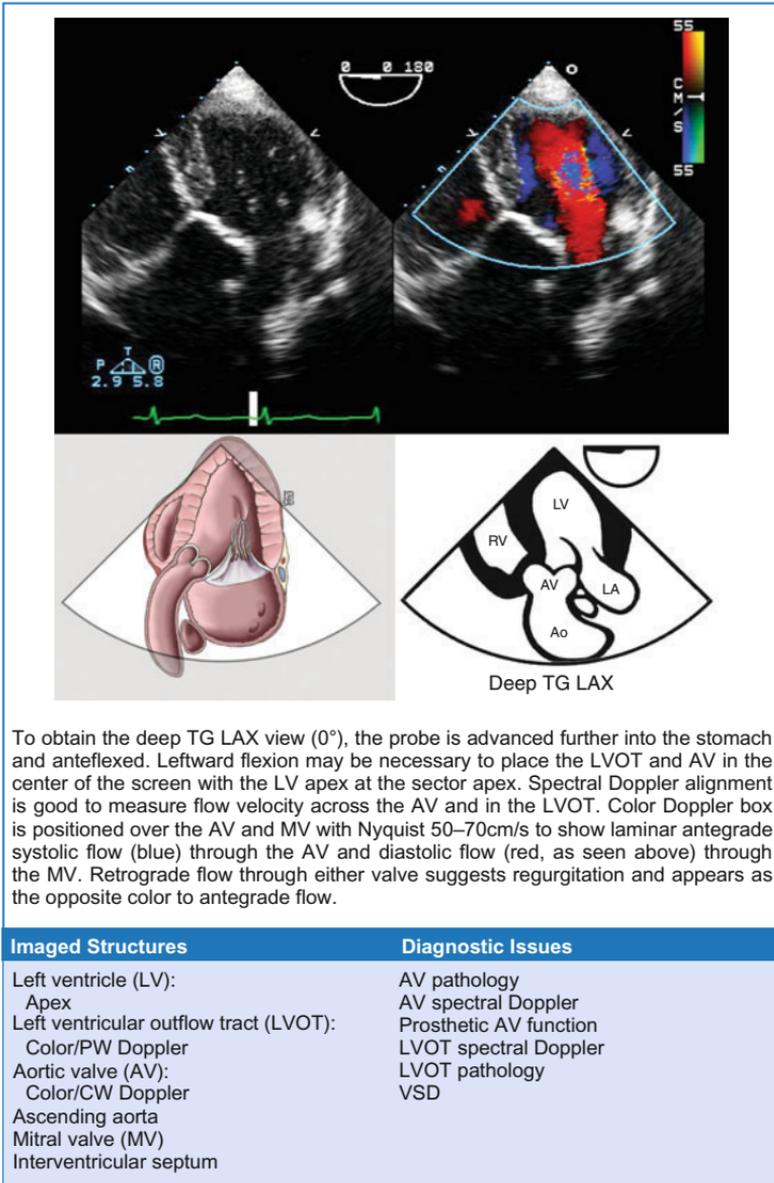
### Imaged Structures

Left ventricle (LV):  
 Antero-septal + posterior walls (basal)  
 Left ventricular outflow tract (LVOT):  
 PW Doppler  
 Interventricular septum (IVS)  
 Mitral valve (MV):  
 Leaflets (anterior and posterior)  
 Subvalvular apparatus  
 Color Doppler  
 Aortic valve (AV):  
 Color/CW Doppler  
 Cusps (RCC is anterior)

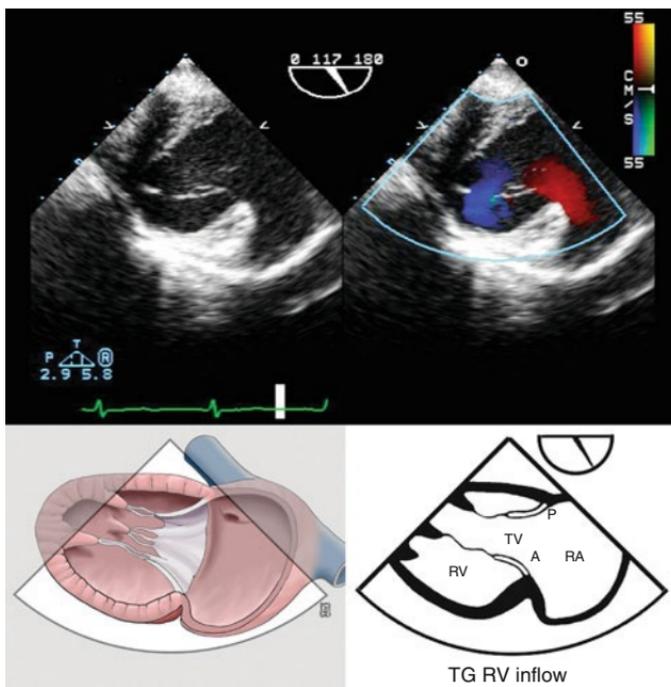
### Diagnostic Issues

MV: leaflets, subvalvular  
 LV systolic function  
 AV Doppler gradient  
 LVOT Doppler gradient  
 Ventricular septal defect (VSD)  
 Prosthetic AV function

## Transgastric Deep Long-Axis (TG Deep LAX)



## Transgastric Right Ventricular Inflow (TG RV Inflow)



The TG RV inflow view ( $90^{\circ}$ – $120^{\circ}$ ) reveals a long axis view of the RV, with the apex of the RV (left) and the anterior free wall in the far field. It is obtained from the TG basal SAX view ( $0^{\circ}$ ), by turning the probe to the right to center the tricuspid valve and increasing the omniplane angle to  $110^{\circ}$ . Color Doppler box is positioned over the TV with Nyquist 50–70cm/s to show laminar antegrade diastolic flow. Continuous and unidirectional diastolic flow, in relation to the probe as shown above, appears red from RA through the TV and blue when filling the RV. Retrograde systolic TV flow appears blue or turbulent and suggests tricuspid regurgitation (TR).

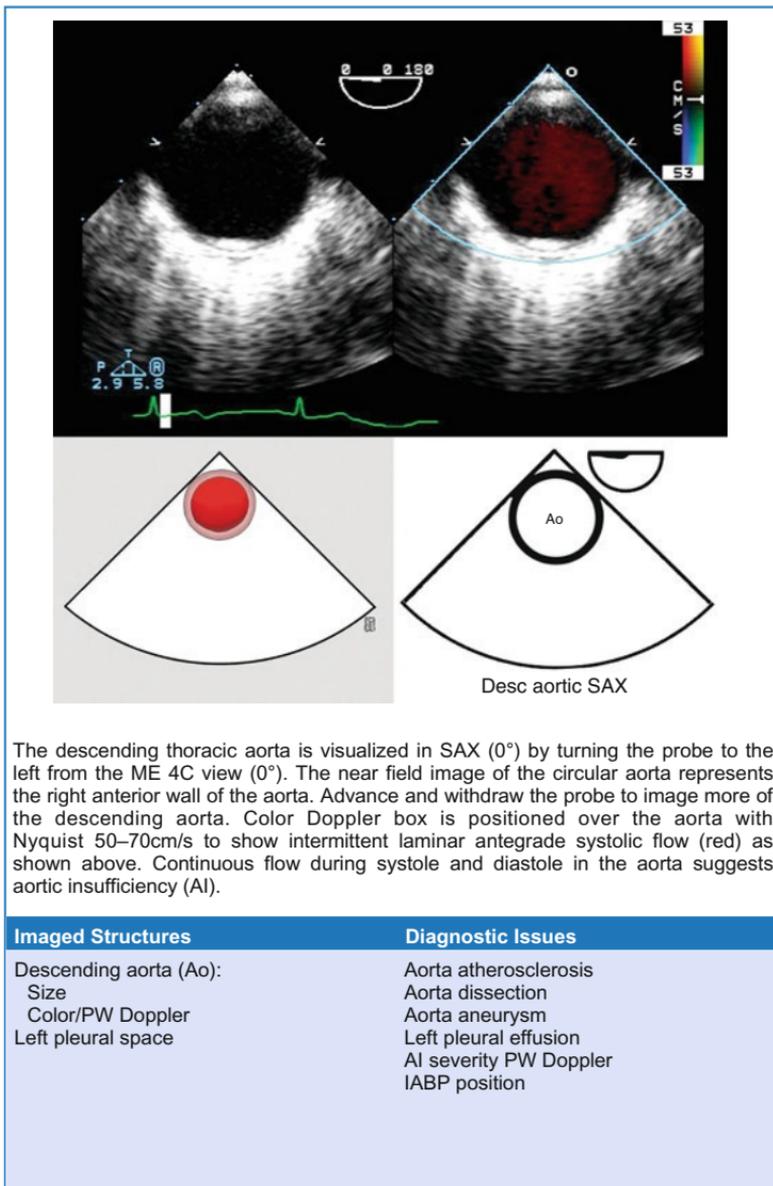
### Imaged Structures

Tricuspid valve (TV):  
 Posterior (P) + anterior (A) leaflets  
 Subvalvular apparatus  
 Color Doppler  
 Right ventricle (RV):  
 Posterior + anterior walls  
 Right atrium (RA)

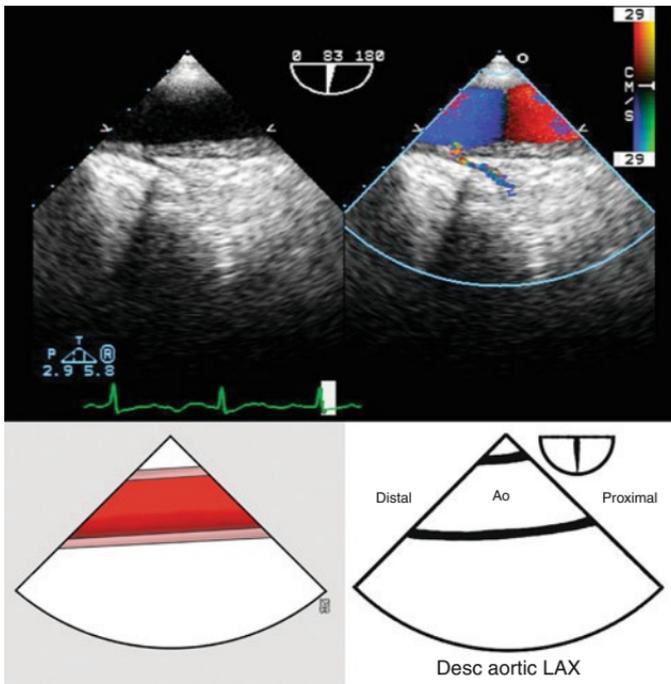
### Diagnostic Issues

TV pathology  
 RV systolic function  
 RA mass

## Mid-esophageal Descending Aortic Short-Axis (SAX)



## Mid-esophageal Descending Aortic Long-Axis (LAX)



From the descending thoracic aortic SAX view ( $0^\circ$ ), the transducer angle is increased to  $90^\circ$  to obtain the Descending Aortic LAX view. The distal aorta is to the display left and the proximal aorta to the display right. Color Doppler box is positioned over the aorta with Nyquist 50–70cm/s to show laminar antegrade systolic flow. Though flow is continuous and unidirectional, in relation to the probe, it appears red through the proximal and blue through the distal descending aorta. Black color indicates the probe is perpendicular to flow. A lower Nyquist limit as shown above can help identify arterial branches at different aortic levels.

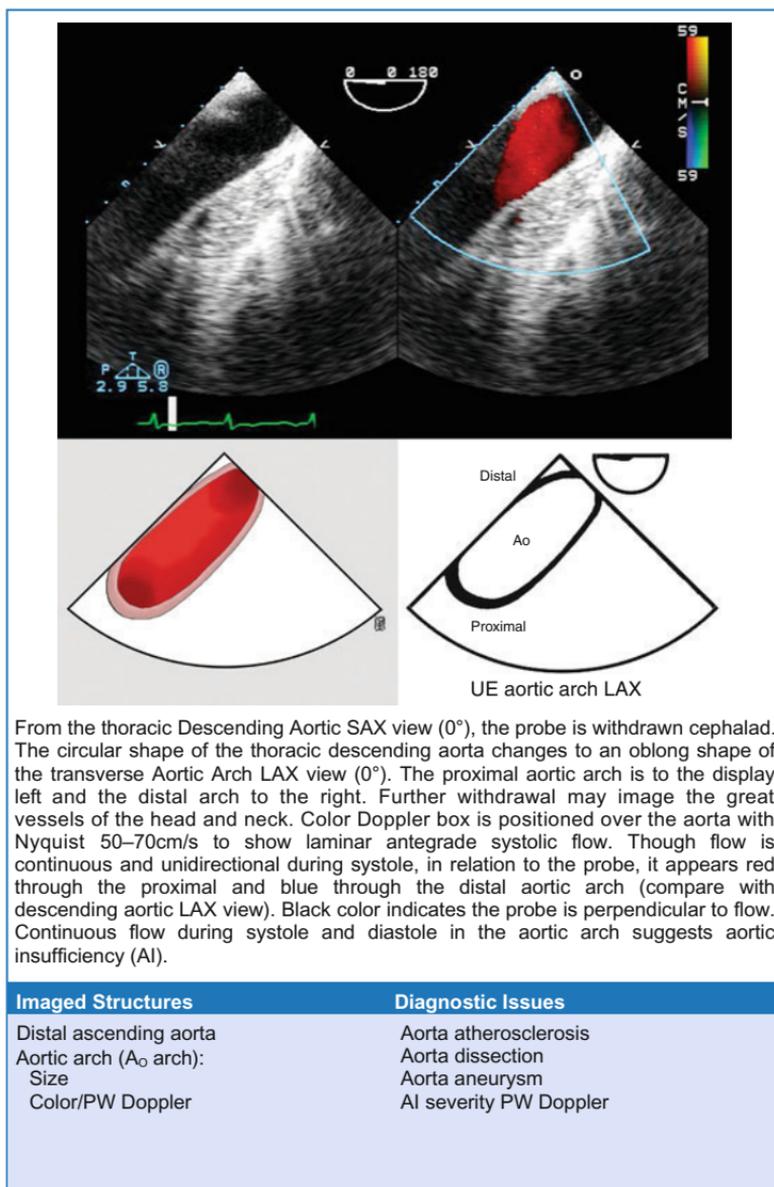
### Imaged Structures

Descending aorta (Ao):  
 Size  
 Color/PW Doppler  
 Intercostal arteries  
 Left pleural space

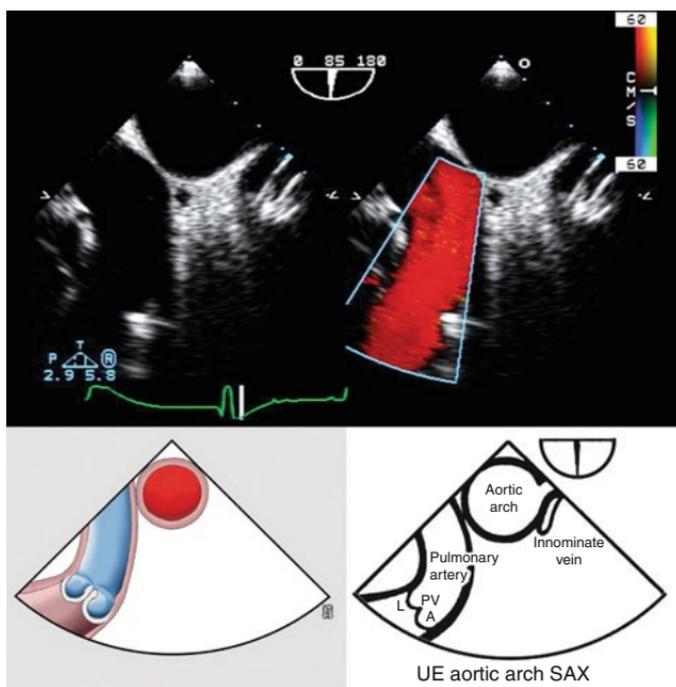
### Diagnostic Issues

Aorta atherosclerosis  
 Aorta dissection  
 Aorta aneurysm  
 AI severity PW Doppler  
 IABP position

## Upper-esophageal Aortic Arch Long-Axis (LAX)



## Upper-esophageal Aortic Arch Short-Axis (SAX)



From the upper esophageal aortic arch LAX view ( $0^\circ$ ), increasing the transducer angle to  $60^\circ$ – $90^\circ$  obtains the UE aortic arch SAX view. This shows the proximal origin of the left subclavian artery and innominate vein in the upper right display. The pulmonic valve (PV) and main pulmonary artery (PA) in LAX are seen in the lower left display. Color Doppler box is positioned over the RVOT, PV, and PA with Nyquist 50–70cm/s to show laminar antegrade systolic flow. Retrograde diastolic flow suggests pulmonic insufficiency. The color Doppler box can be separately positioned over the aortic arch with Nyquist of 70–90cm/s and a lower Nyquist of 30 cm/s over the innominate vein.

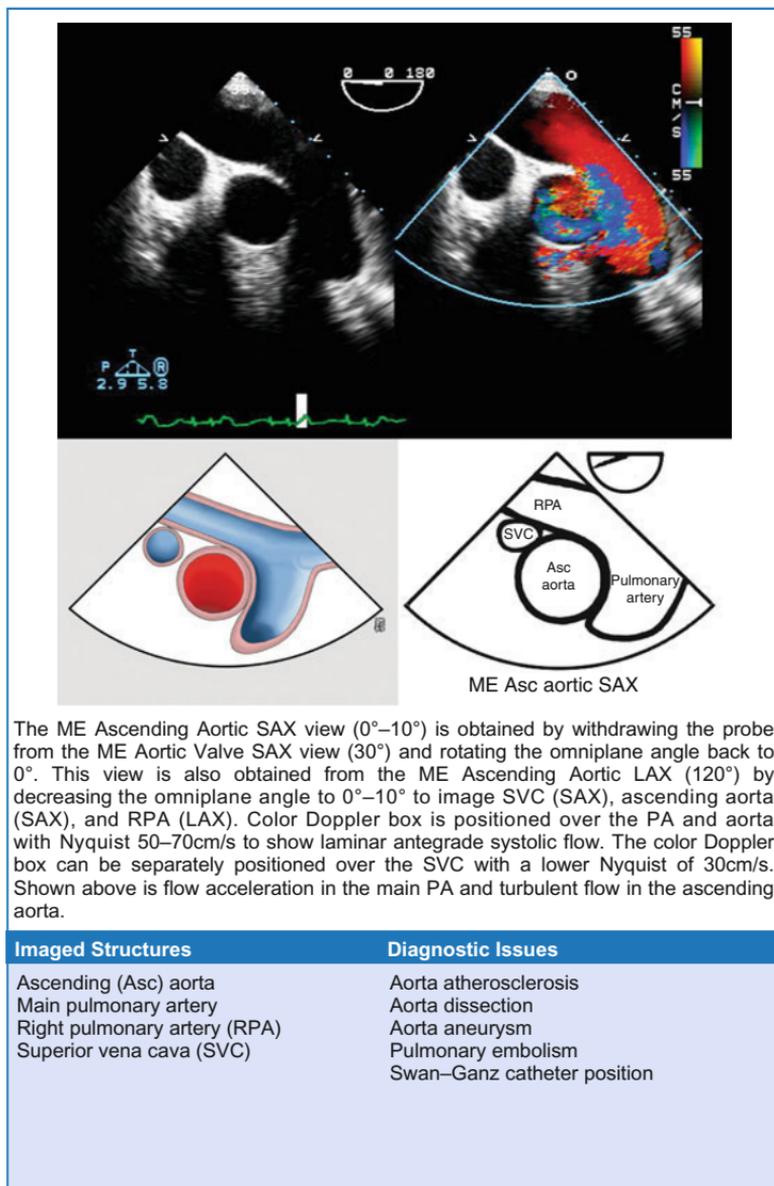
### Imaged Structures

Aortic arch  
 Pulmonary artery (PA):  
 Color/PW Doppler  
 Pulmonic valve (PV):  
 Left (L) and anterior (A) cusps  
 Color/PW Doppler  
 Innominate vein

### Diagnostic Issues

Aorta atherosclerosis  
 Aorta dissection  
 Aorta aneurysm  
 Pulmonic valve pathology  
 Patent ductus arteriosus (PDA)  
 Swan–Ganz catheter position

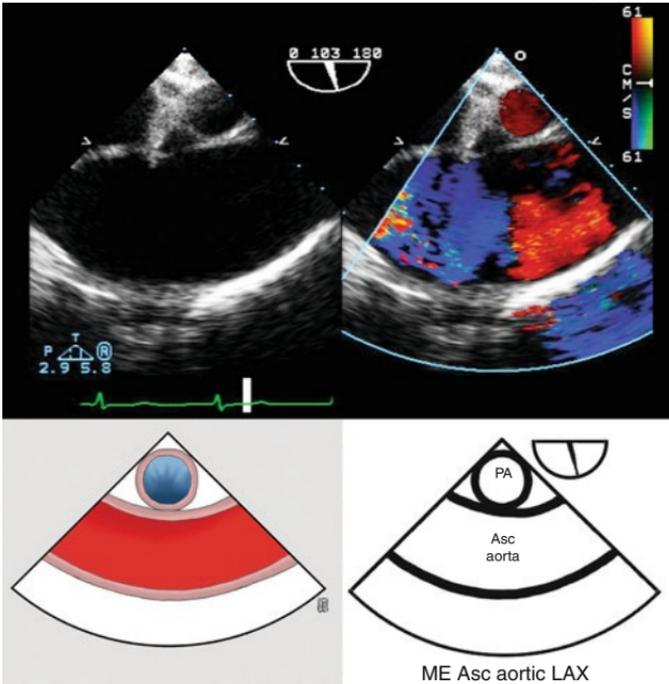
## Mid-esophageal Ascending Aortic Short-Axis (SAX)



The ME Ascending Aortic SAX view ( $0^{\circ}$ – $10^{\circ}$ ) is obtained by withdrawing the probe from the ME Aortic Valve SAX view ( $30^{\circ}$ ) and rotating the omniplane angle back to  $0^{\circ}$ . This view is also obtained from the ME Ascending Aortic LAX ( $120^{\circ}$ ) by decreasing the omniplane angle to  $0^{\circ}$ – $10^{\circ}$  to image SVC (SAX), ascending aorta (SAX), and RPA (LAX). Color Doppler box is positioned over the PA and aorta with Nyquist 50–70cm/s to show laminar antegrade systolic flow. The color Doppler box can be separately positioned over the SVC with a lower Nyquist of 30cm/s. Shown above is flow acceleration in the main PA and turbulent flow in the ascending aorta.

Imaged Structures	Diagnostic Issues
Ascending (Asc) aorta	Aorta atherosclerosis
Main pulmonary artery	Aorta dissection
Right pulmonary artery (RPA)	Aorta aneurysm
Superior vena cava (SVC)	Pulmonary embolism
	Swan–Ganz catheter position

## Mid-esophageal Ascending Aortic Long-Axis (LAX)



The ME Ascending Aortic (LAX) view may be visualized from the ME AV LAX ( $120^\circ$ ), by withdrawing the probe to image the right pulmonary artery (RPA) in SAX, and decreasing the omniplane angle slightly ( $100^\circ$ – $110^\circ$ ). Color Doppler box is positioned over the aorta and RPA with Nyquist 50–70cm/s to show laminar antegrade systolic flow. Though flow is continuous and unidirectional during systole, in relation to the probe, it appears red through the proximal and blue through the distal ascending aorta. In late systole and early diastole, flow is in the opposite direction to facilitate closure of the AV. Black color indicates the probe is perpendicular to flow. Turbulent systolic flow suggests aortic stenosis. Continuous flow during systole and diastole in the aorta indicates aortic insufficiency (AI).

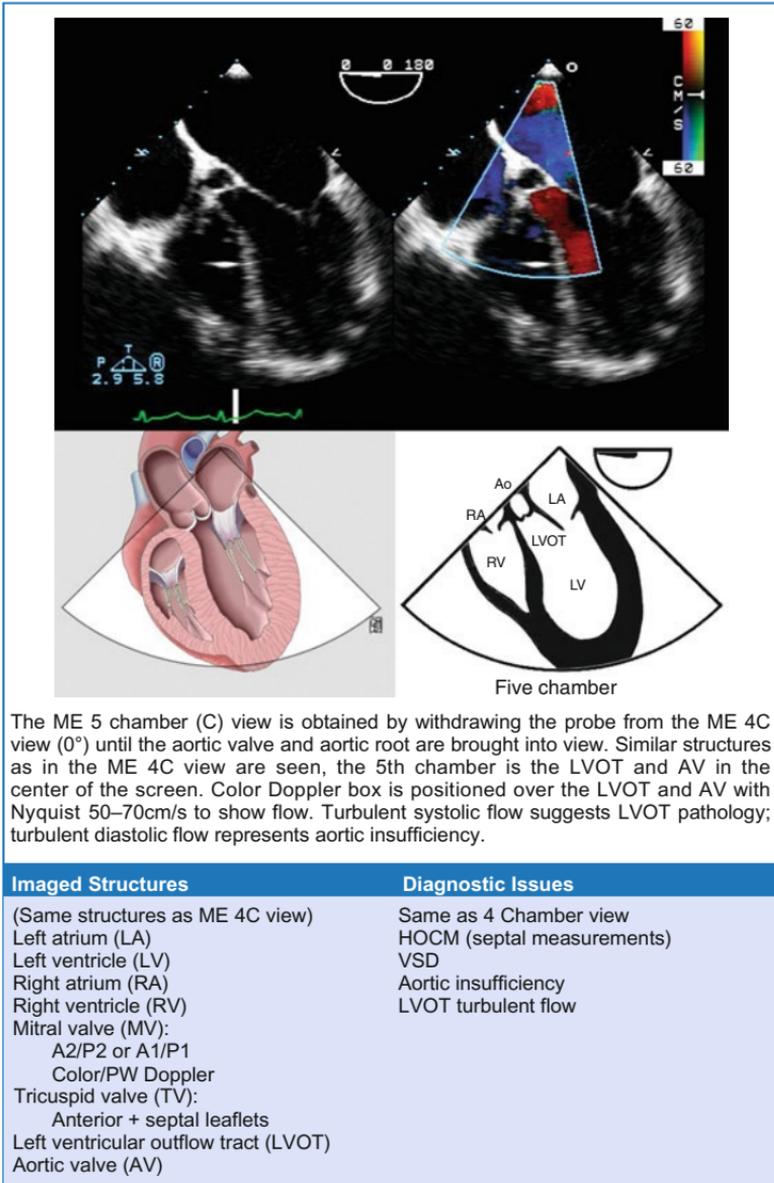
### Imaged structures

Ascending (Asc) aorta:  
Color Doppler  
Right pulmonary artery (RPA)  
Transverse pericardial sinus

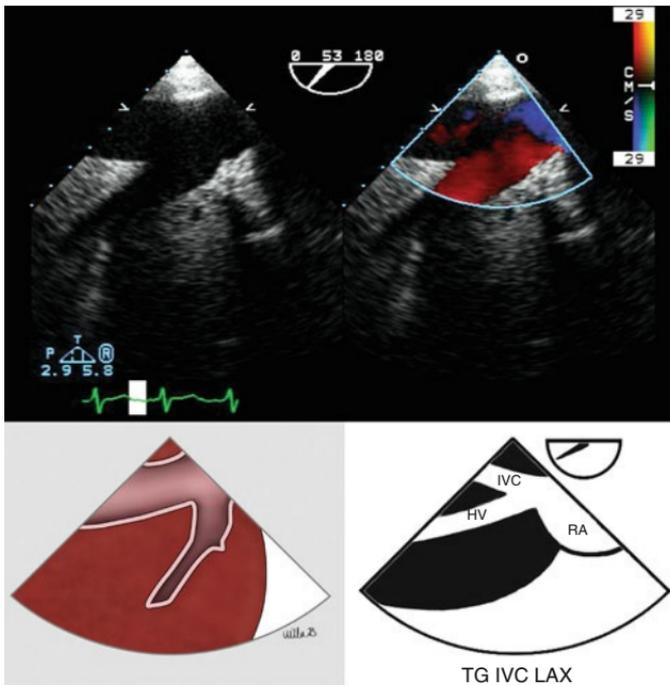
### Diagnostic Issues

Aorta atherosclerosis  
Aorta dissection  
Aorta aneurysm  
Aortic insufficiency flow  
Aortic stenosis flow  
Swan–Ganz catheter in RPA  
Pericardial effusion

## Mid-esophageal Five Chamber (ME 5C)



## Transgastric Inferior Vena Cava (TG IVC)



The transgastric (TG) inferior vena cava (IVC) LAX view is obtained by advancing the probe to image the TG mid SAX view ( $0^\circ$ ). Turn the probe right to find the liver, withdraw to find the IVC as it enters the RA. Adjust the probe and omniplane angle to identify the hepatic vein (HV) as it enters the IVC. Color Doppler box is positioned over the hepatic vein and IVC with a low Nyquist  $30\text{cm/s}$  to show laminar antegrade flow. In relation to the probe, flow appears red through the proximal IVC and hepatic vein and blue into the right atrium (RA).

### Imaged Structures

Inferior vena cava (IVC):  
 Size  $1.6 \pm 0.2\text{cm}$   
 Color Doppler (low velocity)

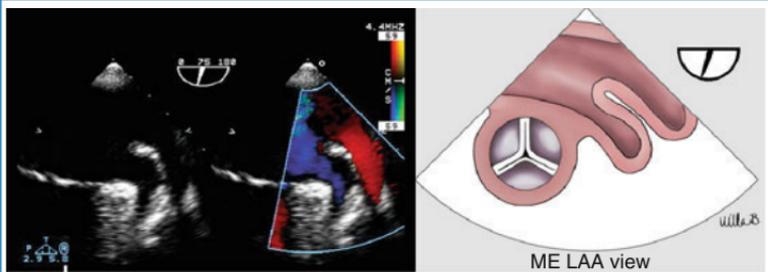
Hepatic vein (HV):  
 Size  $0.8 \pm 0.3\text{cm}$   
 Color Doppler (low velocity)  
 PW Doppler

### Diagnostic Issues

Tricuspid regurgitation  
 Mass (tumor, thrombus)  
 IVC cannula position  
 IVC respiratory variation

## Mid-esophageal Left Atrial Appendage (ME LAA)

### Left Atrial Appendage



The Left Atrial Appendage (LAA) view is obtained by reducing the image depth from the ME RVOT view and adjusting the omniplane angle between 60°–80°. The LAA is seen above the mitral valve or AV with left upper pulmonary vein (LUPV) above (more posterior and closer to the probe). Color Doppler box is positioned over the LAA, and LUPV with Nyquist 50–70cm/s shows laminar flow here during systole.

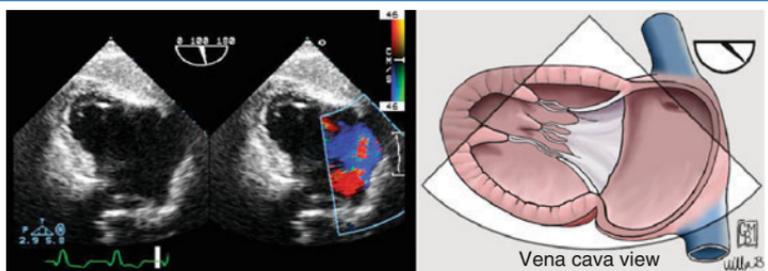
#### Imaged Structures

Left atrial appendage (LAA):  
 Size: diameter  $1.6 \pm 0.5\text{cm}$ , length  $2.9 \pm 0.5\text{cm}$   
 Color/PW Doppler  
 Left upper pulmonary vein (LUPV):  
 Color/PW Doppler  
 Aortic valve (AV)

#### Diagnostic Issues

LAA pathology  
 LUPV flow

### Superior and Inferior Vena Cava



This TG view of both cavae is obtained from the TG RV inflow view by adjusting the omniplane angle and/or rotating the probe slightly. This view gives alignment for spectral Doppler of both vena cavae.

#### Imaged Structures

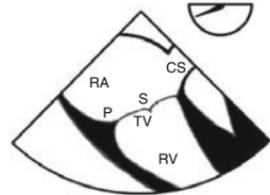
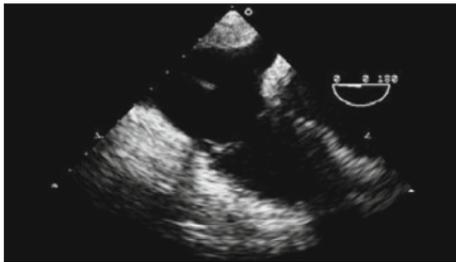
Right atrium (RA)  
 Right ventricle (RV)  
 Tricuspid valve (TV)  
 Inferior vena cava (IVC)  
 Superior vena cava (SVC)

#### Diagnostic Issues

TV pathology  
 IVC PW Doppler  
 SVC PW Doppler

## Mid-esophageal Coronary Sinus

### Coronary Sinus



Coronary sinus

The Coronary Sinus LAX view ( $0^\circ$ ) is obtained at the gastroesophageal junction by advancing the probe from ME 4C view or withdrawing the probe from the TG basal SAX view. The coronary sinus (CS) is seen in LAX entering the RA above the TV.

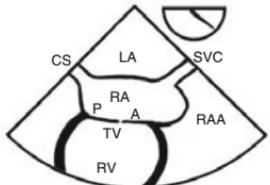
#### Imaged Structures

Right atrium (RA)  
 Right ventricle (RV)  
 Tricuspid valve (TV):  
   Septal (S) + posterior (P) leaflets  
 Coronary sinus (CS):  
   Size: diameter  $0.7 \pm 0.2$ cm

#### Diagnostic Issues

Dilated CS ( $>2$ cm), persistent left SVC  
 TV pathology  
 CS cardioplegia catheter

### Tricuspid Valve



Tricuspid valve

This modified ME view of the tricuspid valve (TV) is obtained from the ME bicaval view by increasing the omniplane angle to  $120^\circ$ – $150^\circ$ . This view gives optimal alignment for spectral Doppler of the TV.

#### Imaged Structures

Right atrium (RA), appendage (RAA)  
 Right ventricle (RV)  
 Tricuspid valve (TV):  
   Anterior + posterior leaflets  
   Color/CW or PW Doppler  
 Coronary sinus (CS)  
 Superior vena cava (SVC)

#### Diagnostic Issues

TV pathology  
 CW Doppler tricuspid regurgitation  
 CS color flow  
 SVC color flow  
 Inter-atrial septum flow

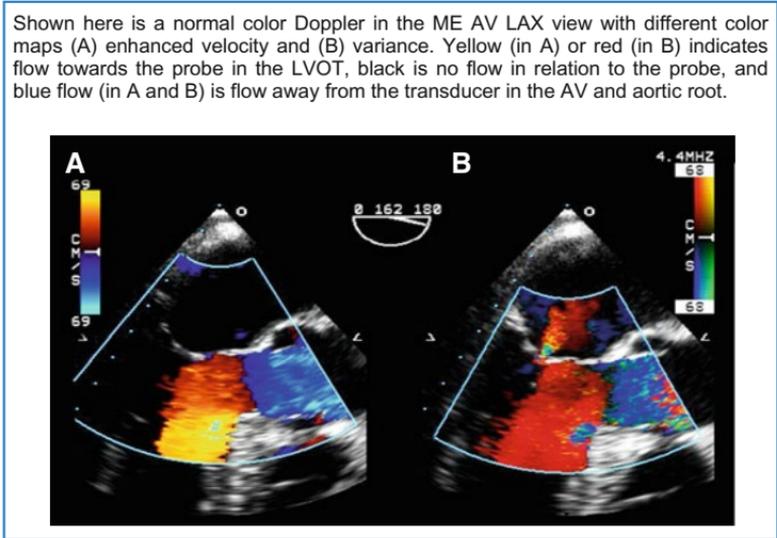
# Doppler and Hemodynamics

Color Doppler .....	32–33
Spectral Doppler .....	34–35
Doppler Artifacts .....	36
Spectral Profiles .....	37–41
Pulmonary Veins .....	42–43
Cardiopulmonary Pressure Estimates .....	44
Aortic Valve: Continuity and Pressure Half-Time .....	45
Mitral Valve Area: Pressure Half-Time and PISA .....	46
Effective Regurgitant Orifice Area .....	47
Regurgitant Volume and Fraction .....	48
Shunt Fraction.....	49

# Color Doppler

## Color Maps

Color Doppler is a form of pulsed Doppler that, after analysis by autocorrelation, displays returning echoes as color superimposed on a 2D image. Large packets of information are analyzed for mean velocity, so unlike spectral Doppler, alignment is not required. By convention, Doppler color flow is assigned a color depending on the direction of flow relative to the transducer: **Blue** is flow **Away**, and **Red** is flow **Towards** (**BART**). The zero velocity baseline in the center is black as there is no frequency shift (no flow). Color Doppler is displayed using different color maps. The enhanced velocity map shows higher velocity flows as brighter colors. Variance color map uses additional colors (yellow and green) to indicate turbulent flow, thus displaying a mosaic of colors. The color scale (or Nyquist limit) can be crudely adjusted. Flow velocity exceeding the Nyquist limit appears aliased (see next page).



- Parameters that can be adjusted in color Doppler mode include:
- Choice of color map: velocity or variance
  - Color scale: changes range of color flow velocities, so crudely adjust Nyquist limits
  - Baseline: shift up/down to change range of color flow velocities in one direction
  - Size and depth of color sector: influences Nyquist limit
  - Color gain: adjusts system sensitivity to received color flow signals (preset 70%)

- Color Doppler assessment examines:
- Anatomical structure (underlying 2D image)
  - Blood flow direction (toward or away from transducer)
  - Mean velocity (frequency shift)
  - Timing with ECG (systole or diastole)
  - Laminar or turbulent flow

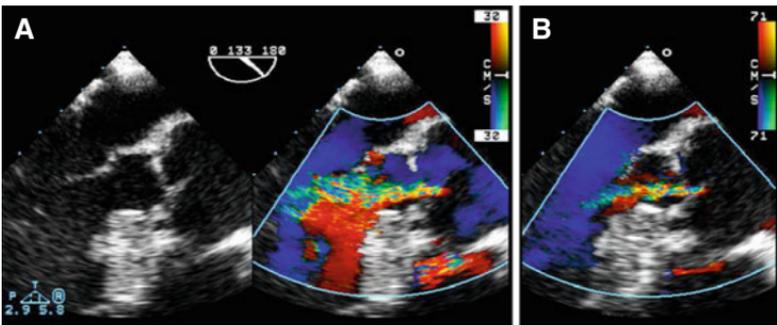
## Color Doppler

### Turbulent Flow

Laminar flow exists when blood flows at the same velocity. Blood flowing at different velocities represents turbulent flow. The difference in mean velocities can be displayed using a variance color map as a mosaic of colors (yellow and green).

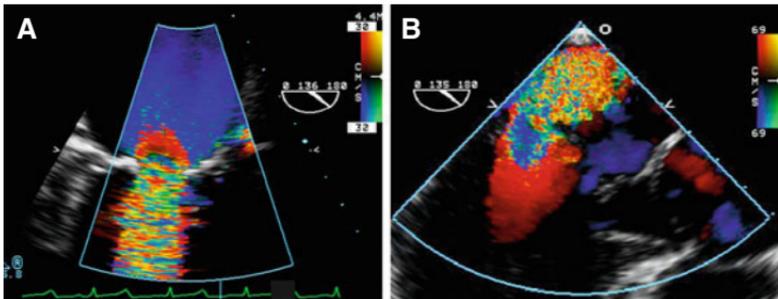
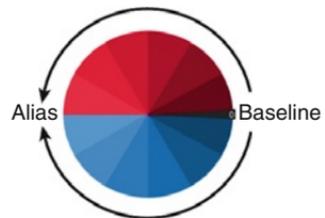
### Color Scale (Nyquist Limit)

To accurately assess the severity of flow velocity, an appropriate Nyquist limit (velocity scale) must be selected: high (aorta), moderate (valves), or low (venous structures). An inappropriately high Nyquist limit may miss flow through a structure, while a low Nyquist limit may overestimate or suggest turbulent flow. In this ME AV LAX view of aortic insufficiency (AI), the (A) low Nyquist limit of 30 cm/s overestimates the AI severity compared with (B), a Nyquist limit of 71 cm/s.



### Aliasing/Flow Acceleration

Aliasing in color Doppler occurs when flow exceeds the Nyquist limit and appears side by side as the opposite color suggesting a change in direction of flow. In reality, flow is still in the same direction. Unlike spectral Doppler, aliasing in color Doppler is a useful means to assess pathology. The presence of flow acceleration within any valve indicates valve pathology, as shown here for mitral (A) stenosis and (B) regurgitation.



# Spectral Doppler

**Doppler Effect:** The frequency of sound emitted from a moving object is shifted in proportion to the velocity of the moving object.

**Doppler Shift (Fd):** Difference in frequencies of source (Ft) and receiver (Fr):

$$F_d = (F_r - F_t)$$

which is (+) when towards and (-) when away. Typical ultrasound Doppler shifts of -10 to 20 KHz are audible.

**Doppler Equation:** Relationship of Doppler shift (Fd) and blood flow velocity (V)

$$V = c(F_d) / 2F_t \cos\theta$$

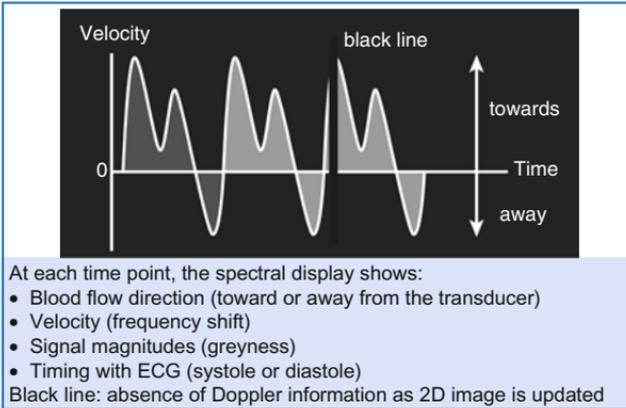
where c is the speed of sound in tissue, Ft is the transducer frequency, and cosθ is the angle between the ultrasound beam and path of moving object.

**Bernoulli Equation:** Relates blood flow velocity to pressure gradient, full equation

$$P_1 - P_2 = 4 (V_1 - V_2)^2 \text{ which if small } V_2 \text{ is simplified to } P_1 - P_2 = 4 V^2.$$

## Spectral Doppler

Returning echoes from moving objects undergo Fast Fourier Transformation to provide a spectral Doppler display showing time (x-axis) and velocity (y-axis). The zero velocity baseline is in the center, frequency shifts toward the transducer above, and frequency shifts away from the transducer below. The amplitude (y-axis) is directly proportional to the measured rbc velocity (Doppler shift). Multiple frequencies exist at any time point, each frequency signal is displayed as a pixel. The magnitude (z-axis) of the Doppler signal is determined by the number of rbcs traveling at each of those velocities and is displayed using various shades of grey. The greyer the display, the more rbcs.



Parameters that can be adjusted in spectral Doppler mode include:

- Scale: adjusts the range of velocities displayed
- Baseline: adjusts the zero baseline velocity up or down
- Doppler gain: alters the overall strength of returning signals
- Grey scale: alters the various ranges of grey displayed
- Wall filter: sets the threshold below which low frequency signals are removed from the display (preset at 500 Hz)
- Sweep speed: changes in ECG rate (25, 50, 100, 150 mm/s) affect Doppler display

## Spectral Doppler

Pulsed Wave Doppler	Continuous Wave Doppler
<ul style="list-style-type: none"> <li>• Uses one crystal in transducer to intermittently send + receive signals</li> <li>• Allows sampling of blood velocity at specific depth (<i>range resolution</i>)</li> <li>• Limit on the maximum velocity seen (aliasing) due to the Nyquist limit (PRF = 2 x transmitted frequency)</li> </ul>	<ul style="list-style-type: none"> <li>• Uses two crystals in transducer to continuously send + receive signals</li> <li>• Sampling occurs along the entire Doppler beam (<i>range ambiguity</i>)</li> <li>• Unlimited maximum velocity displayed (no aliasing), not Nyquist limited</li> </ul>
<p>Source: Quinones MA, et al. J Am Soc Echocardiogr 2002; 15: 167-84</p>	

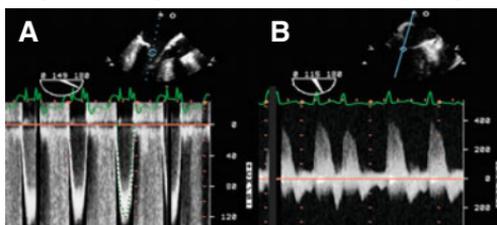
### Velocity

Accurate velocity measurement requires optimal Doppler alignment parallel to blood flow. Different velocities can be measured from the Doppler trace:

- Mean: average velocity obtained by tracing outer edge
- Modal: most common velocity
- Peak: highest velocity

### Aliasing

Aliasing in spectral Doppler occurs when the velocity exceeds the rate at which the pulsed wave Doppler can properly record it. The spectral trace for PW Doppler is cut off and appears to be on the opposite side of the baseline. Shown here is MV inflow using (A) PW with an aliased mitral regurgitation signal that is better displayed by (B) using CW, shifting the baseline down, and adjusting the scale.



### Doppler Indications (Spectral and Color)

Used to diagnose and quantify normal and pathological flows involving:

- Valves: aortic, mitral, pulmonic, and tricuspid
- Great Vessels: SVC, IVC, aorta, PA, hepatic veins, and pulmonary veins
- Defects (ASD, VSD), abnormal connections (fistula, conduits)
- Aortic dissection

## Doppler Artifacts

**Spectral Doppler Artifacts:** Result from abnormal flow or poor Doppler alignment:

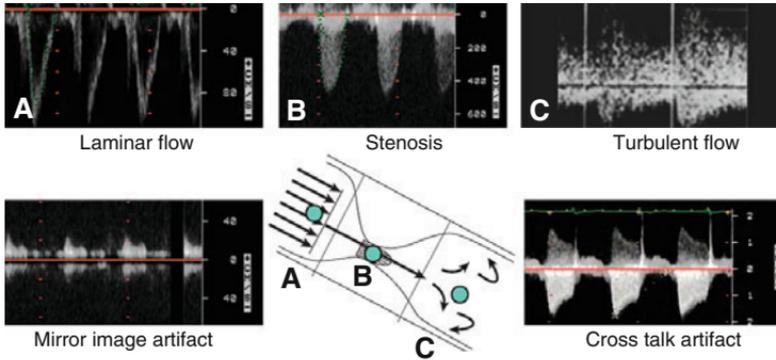
(A) Laminar flow: beam parallel to flow displays optimal trace

(B) Stenosis: high peak velocity usually requires CW Doppler

(C) Turbulent flow: poststenotic area has eddies with different flows

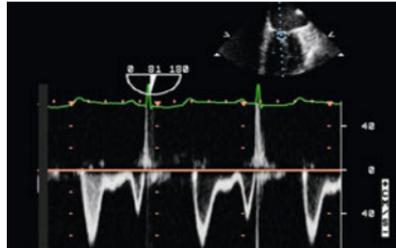
Mirror image artifact: beam perpendicular to flow, identical traces either baseline

Cross talk artifact: high gain, flow both sides of baseline but not identical



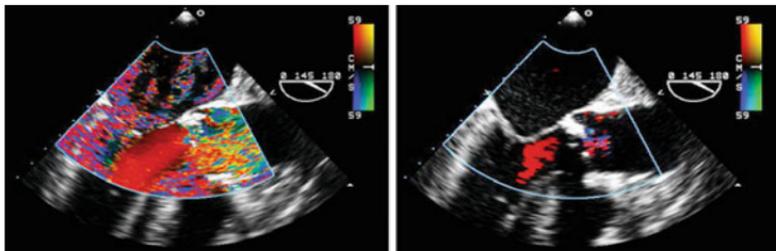
### Spectral Broadening

Implies the presence of a wide range of velocities and appears as a filled in spectral display. While most commonly associated with CW Doppler, it may also occur in PW Doppler when there is irregular flow as in this example of PW MV inflow. In pulmonary vein flow, if the Doppler sample volume is positioned too close to the vessel wall.



### Color Doppler Artifacts

- Shadowing: an absence of color
- Ghosting: brief flashes of color
- Noise: excessive gain (below)
- Absence color: low color gain (below)
- Aliasing: flow acceleration
- Electrical interference: cautery



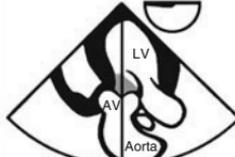
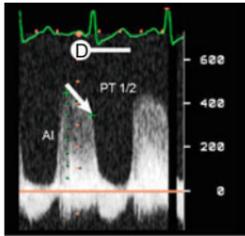
# Spectral Profiles

	<p>TG LAX</p>	<p><b>Aortic Valve</b>            Antegrade            Systolic flow (Ao→LV)            CW Doppler            Flow below baseline            Doppler velocity 1–1.3 m/s            Identify opening and closing valve clicks (arrows)            Rapid rate of acceleration            Diagnostic issues:            Aortic stenosis            Aortic insufficiency</p>
	<p>ME two chamber</p>	<p><b>Mitral Valve</b>            Antegrade            Diastolic flow (LA→LV)            PW Doppler            Leaflet tips or annulus            Flow below baseline            Doppler velocity (&lt;1 m/s)            Emax 0.6–0.8 m/s            Amax 0.2–0.4 m/s            Diagnostic issues:            Diastology            Mitral stenosis            Pericardial tamponade</p>
	<p>UE aortic arch SAX</p>	<p><b>Pulmonic Valve</b>            Antegrade            Systolic flow (RV→PA)            CW Doppler            Flow above baseline            Doppler velocity 0.8–1 m/s            Slower rate of acceleration compared with AV            Diagnostic issues:            Pulmonic stenosis</p>
	<p>ME RV inflow-outflow</p>	<p><b>Tricuspid Valve</b>            Antegrade            Diastolic flow (RA→RV)            PW Doppler            Flow below baseline            Doppler velocity (&lt;0.7 m/s)            Emax <math>0.4 \pm 0.098</math> m/s            Amax <math>0.2 \pm 0.075</math> m/s            respiratory variation so average several cycles            Diagnostic issues:            Tricuspid stenosis            Pericardial tamponade</p>

# Spectral Profiles

	<p>ME four chamber</p>	<p><b>Mitral Regurgitation</b></p> <p>Retrograde (mosaic) Systolic flow (LV→LA) CW Doppler Flow above baseline Doppler velocity 5–6 m/s Signal intensity ∝ MR Estimate LAP = <math>\text{aorticC}_{\text{SBP}} - 4(\text{MR}_{\text{peak}})^2</math></p>
	<p>ME two chamber</p>	<p><b>Mitral Stenosis</b></p> <p>Antegrade (mosaic) Diastolic flow (LA→LV) PW/CW Doppler Flow below baseline Doppler velocity &gt; 3 m/s High mean pressure &gt; 12 mmHg PT1/2 MV area</p>
	<p>ME RV inflow-outflow</p>	<p><b>Tricuspid Regurgitation</b></p> <p>Retrograde (mosaic) Systolic flow (RV→RA) CW Doppler Flow above baseline Doppler velocity &gt; 2.5 m/s Signal intensity ∝ TR Estimate RVSP (PASP) = <math>4 (\text{TR}_{\text{peak}})^2 + \text{RAP}</math></p>
	<p>ME four chamber</p>	<p><b>Tricuspid Stenosis</b></p> <p>Antegrade (mosaic) Diastolic flow (RA→RV) PW/CW Doppler Flow below baseline Doppler velocity &gt; 1.5 m/s Mean pressure gradient &gt; 6 mmHg PT1/2 TV area</p>

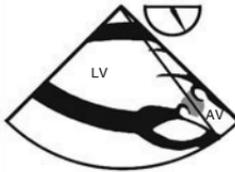
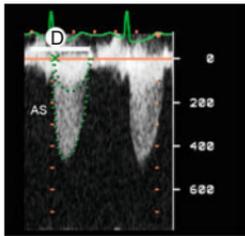
## Spectral Profiles



Deep TG LAX

### Aortic Insufficiency

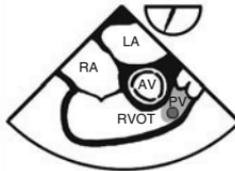
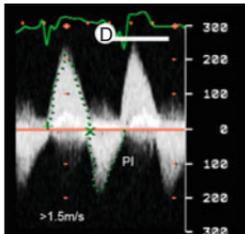
Retrograde (mosaic)  
Diastolic flow (Ao→LV)  
CW Doppler  
Flow above baseline  
Doppler velocity 3–5 m/s  
Signal intensity ∝ AI  
Deceleration slope  
PT1/2  
Estimate LVEDP =  
 $Aortic_{dbp} - 4(AI_{end})^2$



TG LAX

### Aortic Stenosis

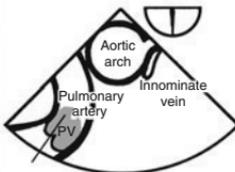
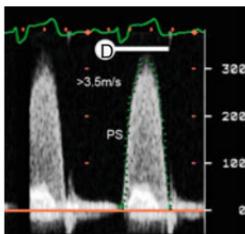
Antegrade (mosaic)  
Systolic flow (LV→Ao)  
CW Doppler  
Flow below baseline  
Doppler velocity > 2 m/s  
Peak/mean P gradients  
VTI for AV area using  
continuity equation



ME RV inflow-outflow

### Pulmonic Insufficiency

Retrograde (blue)  
Diastolic flow (PA→RV)  
PW or CW Doppler  
Flow below baseline  
Doppler velocity > 1.5 m/s  
Signal intensity ∝ PI  
Estimate PADP =  
 $4(PI_{end})^2 + RAP$



UE aortic arch SAX

### Pulmonic Stenosis

Antegrade (red/mosaic)  
Systolic flow (RV→PA)  
PW/CW Doppler  
Flow above baseline  
Doppler velocity > 3.5 m/s  
Peak P grad > 80 mmHg  
VTI for PV area using  
continuity equation

# Spectral Profiles

	<p>Coronary sinus</p>	<p><b>Coronary Sinus</b></p> <p>CS view at GE junction before stomach</p> <p>Flow from CS to RA</p> <p>Laminar flow</p> <p>PW Doppler</p> <p>Doppler below baseline</p> <p>Systolic + diastolic flow</p> <p>Low velocity &lt; 50 cm/s</p> <p>Flow reversal in TR</p>
	<p>ME AV SAX</p>	<p><b>Coronary Artery</b></p> <p>Doppler alignment try: RCA (AV LAX) LCA (AV SAX)</p> <p>PW Doppler</p> <p>Doppler below baseline</p> <p>Systolic(S) + diastolic(D)</p> <p>LMCA (D) <math>71 \pm 19</math> cm/s LMCA (S) <math>36 \pm 11</math> cm/s RMCA (D) <math>39 \pm 12</math> cm/s RMCA (S) <math>25 \pm 8</math> cm/s</p>
	<p>ME asc aortic SAX</p>	<p><b>Pulmonary Artery</b></p> <p>Doppler alignment UE arch SAX or ME asc aortic arch</p> <p>PW Doppler</p> <p>Doppler above baseline</p> <p>Systolic flow</p> <p>Doppler velocity 50 cm/s</p> <p>VTI for cardiac output</p>
	<p>ME two chamber</p>	<p><b>Left Atrial Appendage</b></p> <p>Use ME 2C view</p> <p>PW Doppler LAA</p> <p>Flow pattern depends on rhythm, in NSR: four waves</p> <ol style="list-style-type: none"> <li>1. LAA contraction</li> <li>2. LAA filling, early</li> <li>3. Passive LAA filling, late</li> <li>4. LAA emptying early</li> </ol> <p>Atrial systole <math>60 \pm 8</math> cm/s</p> <p>Atrial diastole <math>52 \pm 13</math> cm/s</p> <p>velocity <math>20 \pm 11</math> cm/s</p>

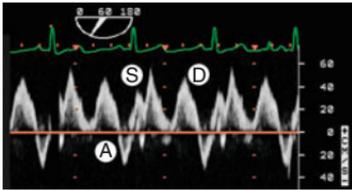
# Spectral Profiles

	<p>Hepatic vein</p>	<p><b>Hepatic Vein</b></p> <p>Four phases:            A: Retrograde flow after atrial contraction            S: Antegrade systolic atrial filling from IVC            V: Retrograde end systolic flow from ventricular contraction            D: Antegrade diastolic flow during RV filling</p> <p>Abnormal flow:            Reverse S: severe TR            Blunted S: RV dysfunction</p>
	<p>TG LAX</p>	<p><b>Ascending Aorta</b></p> <p>Use TG LAX view            Systolic flow            PW Doppler            Doppler below baseline            Max velocity of 1.4 m/s            VTI for continuity equation</p>
	<p>Desc aortic SAX</p>	<p><b>Descending Aorta</b></p> <p>Use Desc Aortic SAX            Systolic flow            PW Doppler            Doppler above baseline            Velocity 100 cm/s (1 m/s)            Diastolic flow reversal indicates aortic insufficiency (AI)            More distal in the descending thoracic aorta the flow reversal, the more severe the AI</p>
	<p>UE aortic arch LAX</p>	<p><b>Distal Aortic Arch</b></p> <p>Use UE Aortic Arch LAX            Systolic flow            PW Doppler            Doppler above baseline            Velocity 100 cm/s (1 m/s)            Diastolic flow reversal indicates aortic insufficiency (AI)</p>

# Pulmonary Veins

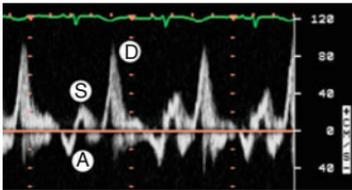
## Pulmonary Vein Flow

- Assess flow in pulmonary veins
- Severity of mitral regurgitation (MR)
- Diastolic function (see pg. 214)



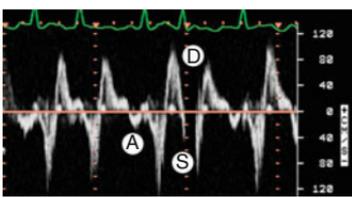
**Normal Flow Pattern: Three phases**  
 A-wave (atrial reversal) 14–25 cm/s  
 S-wave (vent systole) 28–82 cm/s  
 D-wave (vent diastole) 27–72 cm/s  
 Peak S-wave to D-wave ratio  $\geq 1$

- Highly specific for mild MR
- Diastolic function: normal or impaired relaxation



**Blunted Pattern**  
 Peak S-wave to D-wave ratio  $< 1$

- Not specific for moderate MR
- Diastolic function:
  - S < D: pseudonormal
  - S  $\ll$  D: restricted filling



**Systolic Reversal**  
 Ratio of S to D  $< 0$   
 Retrograde flow in mid/late systole

- Highly specific for severe MR

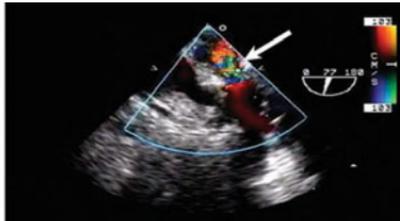
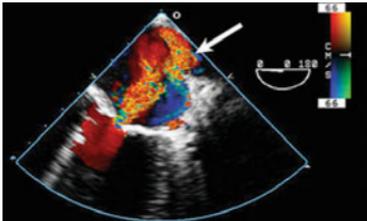
## Turbulent Pulmonary Vein Flow

### Systolic Flow Reversal

Occurs with severe MR, shown here as turbulent retrograde color flow in the LUPV during systole. PW Doppler trace placed 1 cm within the LUPV shows systolic flow reversal (see above).

### Pulmonary Vein Anastomosis

Turbulent antegrade color flow is seen (arrow) within a stenotic pulmonary vein (LUPV) anastomosis during lung transplant. Peak velocity is elevated. Note high velocity color scale and flow acceleration.



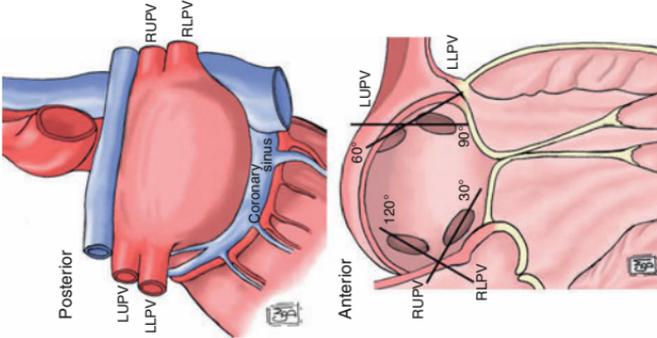
# Pulmonary Veins

## LUPV (ME 60° view)

Easiest pulmonary vein to image. Find left atrial appendage (LAA) in ME 60°, withdraw probe slightly, LUPV lies above (posterolateral) to the LAA and courmadin ridge. Laminar color Doppler flow.

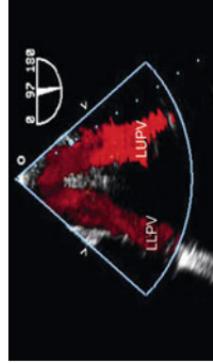


## Pulmonary Vein Anatomy



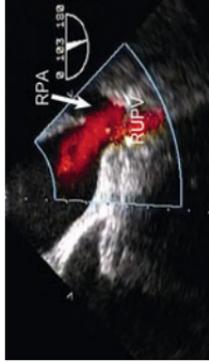
## LLPV + LUPV (ME 90° view)

The LLPV is the most difficult pulmonary vein to image. One technique is to image the LUPV, keeping it in view, increase the omniplane angle to 90°. The left-sided veins appear as an inverted "V".



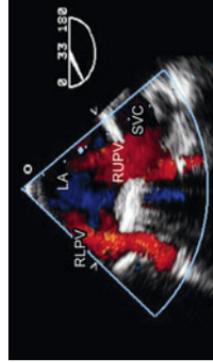
## RUPV (ME 120° view)

The RUPV is easily imaged in the modified ME bicaval view at 110°–120°. From the bicaval view, increase the omniplane angle to 120°, the RUPV appears in the display right near the right pulmonary artery (RPA).



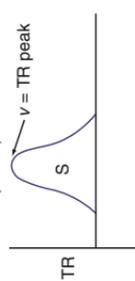
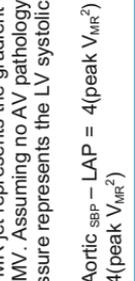
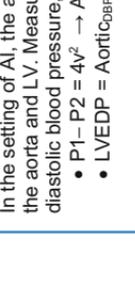
## RLPV + RUPV (ME 30° view)

Turn the probe to image the right side of the LA. The RLPV is imaged from 0° to 30° above (posterior) and perpendicular to the LA. The RUPV is imaged at 30°, below (anterior) to the RLPV.



# Cardiopulmonary Pressure Estimates

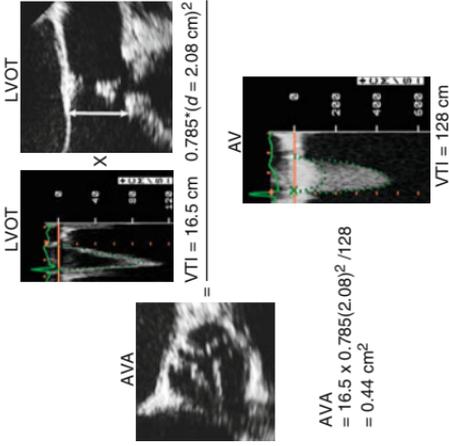
## Estimates of Cardiopulmonary Pressures

<p><b>Tricuspid Regurgitation (TR) to Calculate RVSP (PASP)</b></p> <p>A systolic pressure gradient exists between the RV (P1) and RA (P2), across a closed tricuspid valve. Can estimate the pressure drop from the peak velocity of TR jet.</p> <ul style="list-style-type: none"> <li>• <math>P1 - P2 = 4v^2 \rightarrow P1 = 4v^2 + P2 \rightarrow RVSP = 4(\text{peak TR})^2 + RAP</math></li> </ul> <p>The RAP is estimated from CVP or use an empiric value of 5–10 mmHg. Important clinically as is a noninvasive estimate of PASP that reflects the severity of pulmonary hypertension (in absence of pulmonary stenosis or RVOT obstruction).</p>	 <p>RVSP (PASP) = <math>4v^2 + RAP</math></p> <p>TR</p>
<p><b>Pulmonic Insufficiency (PI) to Calculate PA Diastolic Pressure (PADP)</b></p> <p>In the setting of PI, the pulmonic valve is closed and a pressure gradient exists across the PA and RV. Measure the end diastolic PI velocity. This provides the pressure gradient between the PA and RV at end diastole and by adding RAP can estimate PADP.</p> <ul style="list-style-type: none"> <li>• <math>PADP = 4 \times (\text{PI end diastolic velocity})^2 + RAP</math></li> <li>• Mean PAP = <math>4 \times (\text{PI peak velocity})^2</math></li> </ul>	 <p>PADP = <math>4v^2 + RAP</math></p> <p>PI</p>
<p><b>Mitral Regurgitation (MR) to Calculate LAP</b></p> <p>The peak velocity of the MR jet represents the gradient between the LV and LA during systole across a closed MV. Assuming no AV pathology or LVOT obstruction, the aortic systolic blood pressure represents the LV systolic pressure and can be used to calculate the LAP.</p> <ul style="list-style-type: none"> <li>• <math>P1 - P2 = 4v^2 \rightarrow \text{Aortic SBP} - \text{LAP} = 4(\text{peak } V_{MR})^2</math></li> <li>• <math>\text{LAP} = \text{Aortic SBP} - 4(\text{peak } V_{MR})^2</math></li> </ul>	 <p>LAP = <math>\text{Aortic SBP} - 4v^2</math></p> <p>MR</p>
<p><b>Aortic Insufficiency (AI) to Calculate LVEDP</b></p> <p>In the setting of AI, the aortic valve is closed and a pressure gradient exists between the aorta and LV. Measure the end diastolic AI velocity peak. Knowing the aortic diastolic blood pressure, use the peak end diastolic AI velocity to estimate LVEDP.</p> <ul style="list-style-type: none"> <li>• <math>P1 - P2 = 4v^2 \rightarrow \text{Aortic DBP} - \text{LVEDP} = 4(\text{AI}_{\text{end}} V)^2</math></li> <li>• <math>\text{LVEDP} = \text{Aortic DBP} - 4(\text{AI}_{\text{end}} V)^2</math></li> </ul>	 <p>LVEDP = <math>\text{Aortic DBP} - 4v^2</math></p> <p>AI</p>

# Aortic Valve: Continuity and Pressure Half-Time

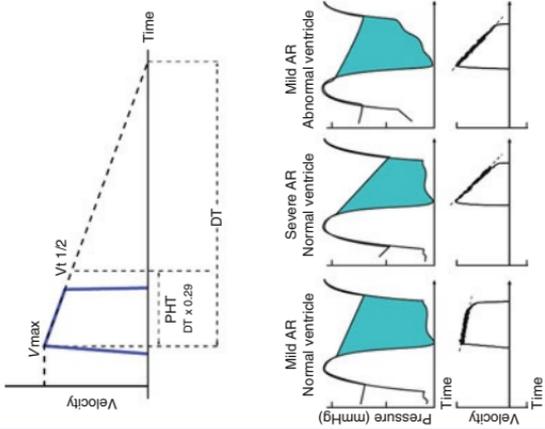
## Aortic Valve Area (AVA) Continuity Equation

$$A_2 = \frac{V_1 \times A_1}{V_2} = \frac{VTI_{LVOT} \times 0.785 d^2_{LVOT}}{VTI_{AV}} = AVA$$



## Aortic Valve Pressure Half-Time (PHT or PT1/2)

The rate of regurgitant velocity decline is determined by the pressure difference between the aorta and LV. The worse the regurgitation (wider orifice), the more rapid decline of aortic and rise in LV pressures causing a steeper slope in regurgitant velocity decline. The diastolic decay slope is measured as the deceleration slope (m/s) or the time (milliseconds) = or the time (milliseconds) for the pressure gradient to fall to half of its initial value, the pressure half time (PT1/2 or PHT). The deceleration time (DT) is the time for Vmax to fall to 0 and relates to PHT by 0.07Vmax. Both ↑ SVR and ↓ LV compliance affect the regurgitant velocity slope irrespective of the regurgitant orifice size.

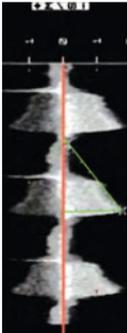


- In theory can use ratio of peak velocities, mean velocities, or VTI multiplied by LVOT CSA to calculate AVA.
- Use ME AV LAX (120°) view to obtain LVOT "d" measurement in mid-systole just below aortic valve (not at AV annulus). Calculate area:  $A = \pi(d/2)^2 = 0.785d^2$ . Can estimate LVOT area as 2.0cm<sup>2</sup> (±0.2 cm<sup>2</sup>)
- Use TG LAX (120°) view, place CW sample cursor through LVOT(AV/root), trace to obtain the VTI.
- Use TG LAX (120°) view, place PW sample at the LVOT level (below valve), trace the spectral envelope to obtain the VTI. Start PW in AV outflow and back towards LVOT until we get a smooth well-defined apex velocity trace and little spectral broadening.

# Mitral Valve Area: Pressure Half-Time and PISA

## Mitral Valve Area (MVA) in Mitral Stenosis

### Pressure Half-Time (PT/1/2, PHT) Method

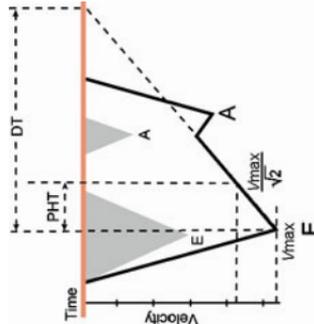


Max velocity (m/s)  
Time (s)  
Max pressure (mmHg)  
PHT (ms)  
MVA (cm<sup>2</sup>)

1. Optimize PW/CW MV inflow (at leaflet tips)
2. Increase sweep speed to 100 m/s
3. Press analysis → MVA PHT<sub>1/2</sub> → dec slope
  - Caliper button: place caliper at peak of E wave + enter
  - Caliper at baseline on the slope of E wave + enter
  - Displays a calculated MVA

In MS, the LAP is elevated as the LA empties slowly through the narrowed MV. CW Doppler shows a decrease or flattening of the deceleration slope (after the E wave). The time it takes for peak pressure to decrease in half is the pressure half-time (PHT). The MVA in native valve MS can be obtained by using the empirical formula:

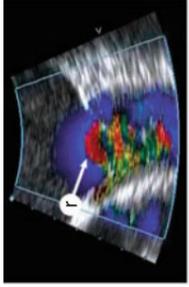
$$MVA = 220 \frac{PHT}{PHT}$$



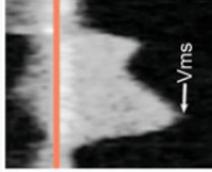
### Proximal Isovelocity Area (PISA) Method

$$MV \text{ Area (cm}^2\text{)} = \text{Flow (cc/s)} \div V_{ms} \text{ (cm/s)}$$

$$\text{Flow rate (cc/s)} = 2 \pi r^2 \times \text{angle } \alpha \times V_{\text{alias}} \div 180^\circ$$



- 2  $\pi r^2$  = hemisphere area
- r (cm) = radius of flow convergence region
- $V_{\text{alias}}$  (cm/s) = aliasing velocity
- angle  $\alpha$  = conversion factor  $\div 180^\circ$



$V_{ms}$  is the peak PW/CW Doppler velocity of MV inflow in cm/s

### Advantages

1. Use if MS or prosthetic valve
2. Use if AI or MR

- Underestimate: post balloon valvuloplasty, impaired relaxation
  - Overestimate: AI, high CO, MR, tachycardia, restrictive filling
  - Unreliable: prosthetic valves, AV block
- Note: In a normal native MV, the PHT is primarily a function of LV compliance and is not useful for estimation of MVA.

# Effective Regurgitant Orifice Area

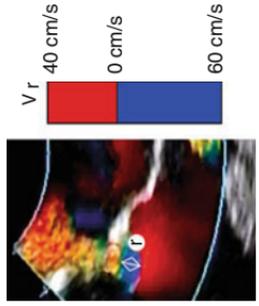
## Effective Regurgitant Orifice (ERO) Area Mitral Regurgitation

EROA represents unique information for valvular regurgitant lesions and correlates to the severity of regurgitation.

- Grade 1: < 1.0 cm<sup>2</sup>
- Grade 2: 1.0 –2.5 cm<sup>2</sup>
- Grade 3-4: 2.5–3.5 cm<sup>2</sup>

Simplified PISA calculation MV EROA:

**PISA for pinheads**  
 $2\pi r^2 \times V_r \propto 180$   
 Assume:  
 MR peak velocity = 400 cm/s  
 $V_r$  at 40 cm/s  
 Then:  
 $ERO \propto r^2/2$



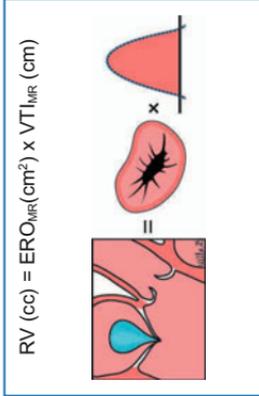
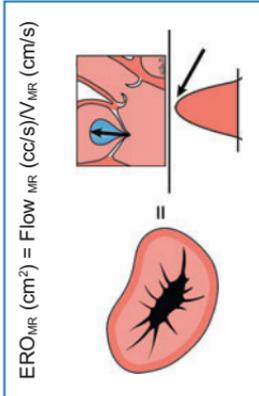
- To measure MR ERO area:
1. Optimize 2D image of MV orifice (zoom)
  2. Optimize color of convergence region
  3. Shift color flow baseline to ↓ the color flow aliasing velocity
  4. Note alias velocity ( $V_r$ )
  5. Using calipers, measure "r" from aliased region to orifice (cm), midsystole
  6. Measure peak regurgitant velocity ( $V_{MR}$ ) of CW jet and trace for VTI

7. Calculate flow MR

$$\text{Flow MR (cc/sec)} = 6.28r^2 \times V_r \text{ (cm/s)}$$

8. Calculate ERO<sub>MR</sub>

9. Calculate regurgitant volume



- More accurate for:
  - Central jets
  - Circular orifice
- Less useful eccentric jets
- Not useful in multiple jets
- Errors in "r" are squared

# Regurgitant Volume and Fraction

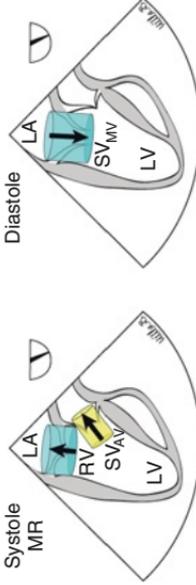
**Regurgitant Volume (RegV):** Volume of blood that regurgitates through an incompetent valve, difference between stroke volume (SV) through incompetent valve and competent valve. Calculate using PISA (see previous page) or continuity equation (below).  
**Regurgitant Fraction (RF):** Fraction or percentage of total stroke volume that regurgitates through an incompetent valve.

$$\text{RegV (cc)} = \text{SV}_{\text{MV}} - \text{SV}_{\text{AV}} \text{ (cc)}$$

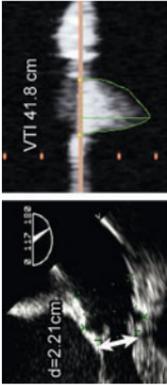
$$\text{RF (\%)} = \frac{\text{SV}_{\text{MV}} - \text{SV}_{\text{AV}}}{\text{SV}_{\text{MV}}} = \frac{\text{RegV}_{\text{MV}}}{\text{SV}_{\text{MV}}}$$

Mild < 40 cc  
 Moderate 40–60 cc  
 Severe > 60 cc

Normal/trivial < 20%  
 Mild 20–30%  
 Moderate 30–50%  
 Severe > 50%



1.  $\text{SV}_{\text{AV}} = \text{VTI}_{\text{AV}} \text{ (cm)} \times \text{CSA} \text{ (cm}^2\text{)}$   
 diameter AV annulus (cm) in ME AV LAX (120°) or planimeter in AV SAX through the aortic outflow (TG views) and trace for stroke distance (cm)
2. Multiplying the two together will give the stroke volume through the AV.

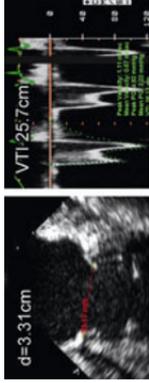


$\text{SV}_{\text{AV}} = 160\text{cc}$

- Normal values
- AV diameter = 1.8–2.2 cm
  - MV diameter = 3.0–3.5 cm
  - VTI AV (CW) = 18–22 cm
  - VTI MV (PW) = 10–13 cm
- Pitfalls:
- PW sample volume location: MV annulus/ LVOT
  - Diameter measurements: location, timing, error is squared
  - Arrhythmias: average 10 beats
  - Multivalve lesions/shunts: formulas invalid if significant shunt or more than mild regurgitation of non-measured valve

1.  $\text{CSA MV} = \pi r^2 = 0.785d^2$  measure diameter MV annulus (cm) at mid-diastole in ME 2C/4C/LAX. Inner to inner edge.
2. VTI MV by obtaining a PW trace at mitral annulus (ME views) and trace for stroke distance (cm).
3. Multiplying the two together will give the stroke volume through the MV.

$$\text{SV}_{\text{MV}} = \text{VTI}_{\text{MV}} \text{ (cm)} \times \text{CSA} \text{ (cm}^2\text{)}$$



$\text{SV}_{\text{MV}} = 221\text{cc}$

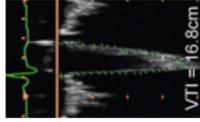
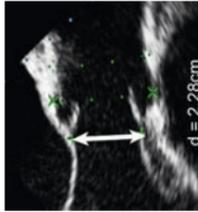
# Shunt Fraction

## Shunt Fractions

The volume of blood flow (Q) across an intra-cardiac shunt compares the stroke volume (SV) at two intra-cardiac sites.

$$SV_{LVOT} = VTI_{LVOT} (cm) \times CSA (cm^2)$$

1. CSA LVOT =  $\pi r^2 = 0.785d^2$  measure diameter LVOT (cm) in ME AV LAX (120°) within 1 cm of AV annulus
2. VTI LVOT by obtaining a PW trace at the aortic outflow (TG views) and trace for (VTI) stroke distance (cm)
3. Multiplying the two together will give the stroke volume through the LVOT



$$SV_{LVOT} = (2.28)^2 \times 0.785 \times 16.8 = 68.5cc$$

### Pitfalls stroke volume:

- Needs accurate x-sectional area measurement
- Assumes laminar flow
- Spatially "flat" flow velocity profile
- Doppler alignment parallel
- Velocity and diameter measurement made at same anatomic site

### Shunt Ratio (ASD)

$$Qp = CSA \times VTI_{PA}$$

$$Qs = CSA \times VTI_{LVOT}$$

Qp = trans-pulmonic volume flow

Qs = LVOT volume flow

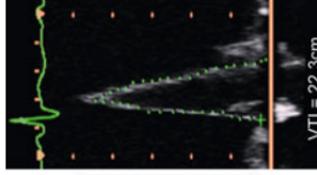
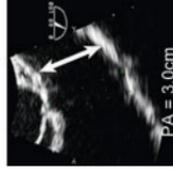
CSA LVOT, VTI LVOT (PW)

CSA PA, VTI PA (PW)

Significant shunt > 1.5:1

$$SV_{PA} = VTI_{PA} (cm) \times CSA (cm^2)$$

1. CSA PA =  $\pi r^2 = 0.785d^2$  measure PA diameter in ME RVOT view
2. VTI PA by obtaining a PW trace through the PA and trace for (VTI) stroke distance (cm)
3. Multiplying the two together will give the stroke volume through the PA



$$SV_{PA} = (3.0)^2 \times 0.785 \times 22.3 = 157.5 cc$$

$$\text{Shunt fraction (Qp/Qs)} = \frac{157.5}{68.5} = 2.3:1$$

### Sample sites for shunts

Shunts	Qp	Qs
	Site distal to shunt inflow	Site distal to shunt outflow
ASD: LA → RA	TV annulus RVOT Main PA	MV annulus LVOT Asc aorta
VSD: LV → RV	RVOT Main PA MV annulus	LVOT Asc aorta TV annulus
PDA: Aorta → PA	MV annulus LVOT Asc aorta	RV annulus TV annulus Main PA

# Ventricles

LV Models .....	52–55
Ventricular Dimensions .....	56–61
Ventricular Wall Motion .....	62
Stroke Volume .....	63
Fractional Area Change and Shortening .....	64
Ejection Fraction .....	65
Ventricular Strain .....	66
Speckle Tracking .....	67
Right Ventricular Function .....	68–69
Coronary Anatomy .....	70
Ischemic Complications .....	71–75

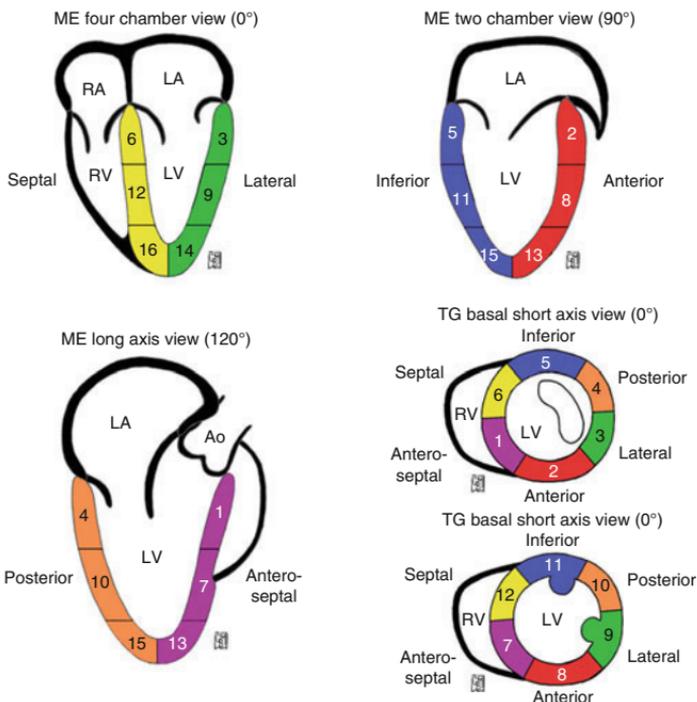
## LV Models

SCA/ASE 16 Segment LV Model		
<b>Basal Segments</b>	<b>Mid Segments</b>	<b>Apical Segments</b>
1. Basal antero-septal	7. Mid antero-septal	13. Apical anterior
2. Basal anterior	8. Mid anterior	14. Apical lateral
3. Basal lateral	9. Mid lateral	15. Apical inferior
4. Basal posterior	10. Mid posterior	16. Apical septal
5. Basal inferior	11. Mid inferior	
6. Basal septal	12. Mid septal	

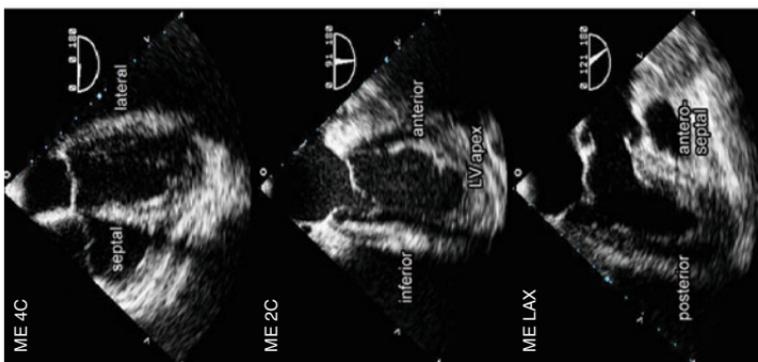
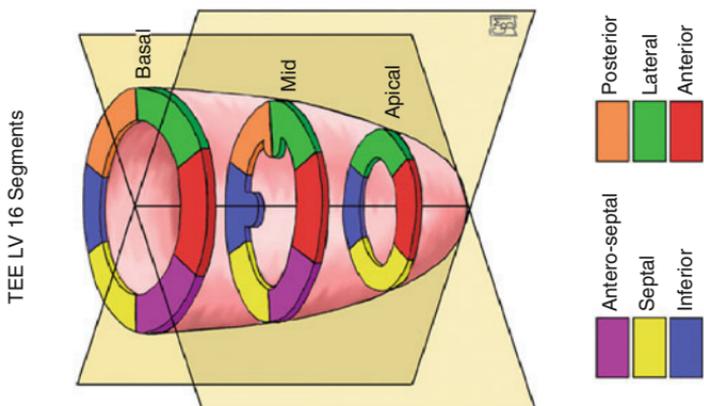
The 16 segment LV model was introduced in 1989 by the ASE and adopted by the SCA in 1996. It is designed to reflect LV regional wall motion and does not include a true apical segment devoid of cavity.

Source: Schiller NB, et al. *J Am Soc Echocardiogr* 1989; 2:358-87.

All 16 LV segments are imaged using both TG SAX and ME views. The TG SAX views image the LV at different TG levels (basal, mid papillary and apex) by advancing and withdrawing the probe in the stomach. Compare the TG and ME LV views for global and regional wall motion abnormalities. Avoid foreshortening the ME LV views by retroflexing the probe tip.



## LV Models



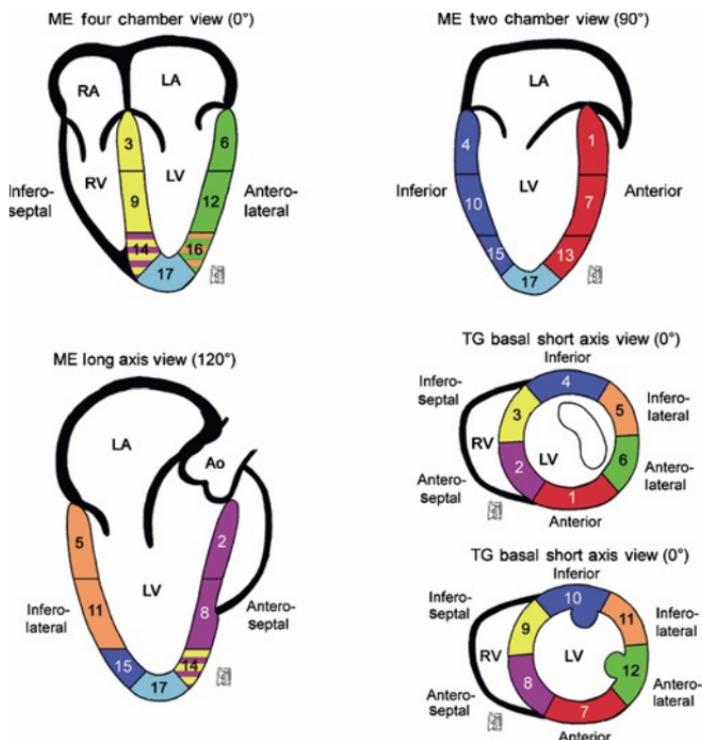
## LV Models

AHA 17 Segment LV Model		
<b>Basal Segments</b>	<b>Mid Segments</b>	<b>Apical Segments</b>
1. Basal anterior	7. Mid anterior	13. Apical anterior
2. Basal antero-septal	8. Mid antero-septal	14. Apical septal
3. Basal infero-septal	9. Mid infero-septal	15. Apical inferior
4. Basal inferior	10. Mid inferior	16. Apical lateral
5. Basal infero-lateral	11. Mid infero-lateral	17. Apex
6. Basal antero-lateral	12. Mid antero-lateral	

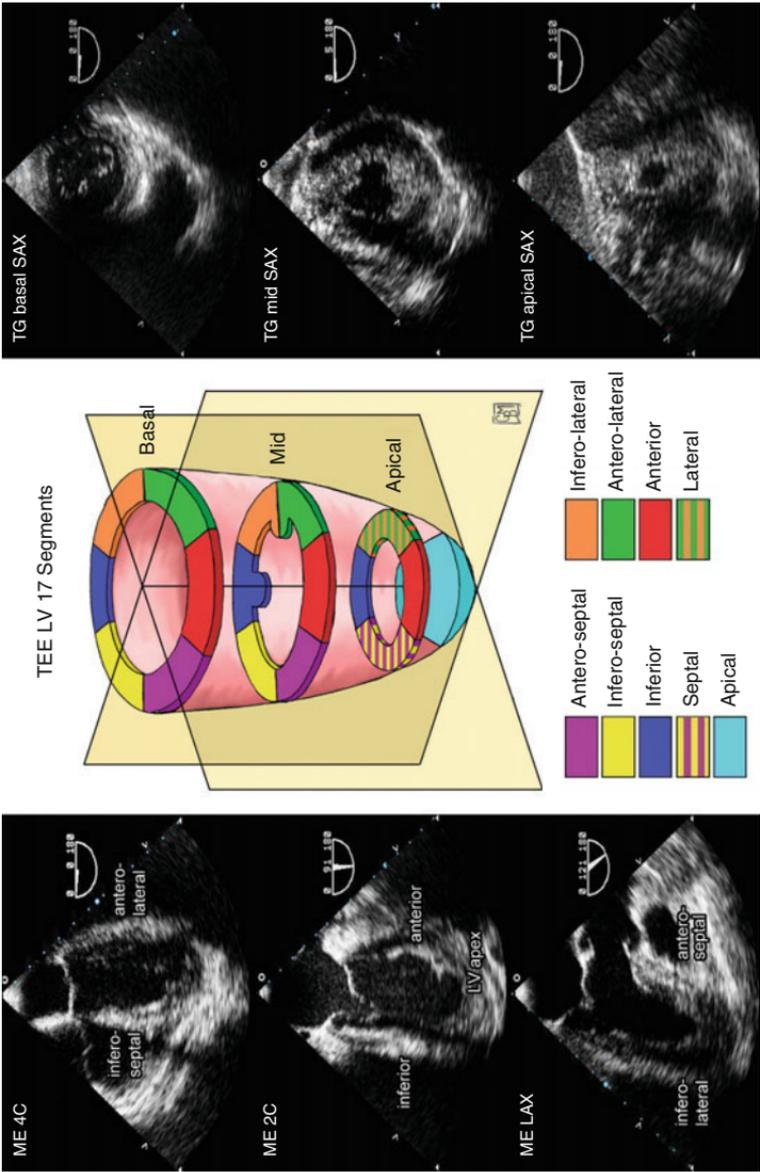
The 17 segment LV model was created in 2002 by the AHA as a consensus guideline to describe LV segmental anatomy for all cardiac imaging modalities. It is used to describe LV regional wall motion and includes a true apical segment devoid of cavity.

Source: Cerqueira M, et al. *Circulation* 2002;105:539-42.

Similar to the 16 segment LV model (see pg. 52), all LV segments are imaged using both the TG SAX and ME views.



# LV Models



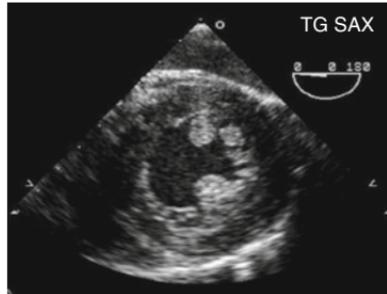
## Ventricular Dimensions

### Left Ventricle Size

LV wall thickness (EDD measurement):  
 Normal LV wall thickness < 12 mm  
 LV Hypertrophy (LVH) > 12 mm

### Differential Symmetric LVH

Hypertension  
 Aortic stenosis  
 Infiltration (amyloid, sarcoid, Fabry's)  
 Metabolic (Cushing, diabetes)  
 Renal disease  
 Athletic heart, obesity  
 Congenital (Noonan, Friedrich's Ataxia)



Normal LV dimensions vary depending on the time of the cardiac cycle and sex of the patient. TEE LV dimensions at end diastole (EDD) have been standardized using the ME and TG 2C views (see pg. 61). Upper limits of LV EDD:

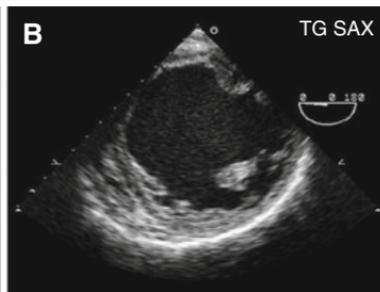
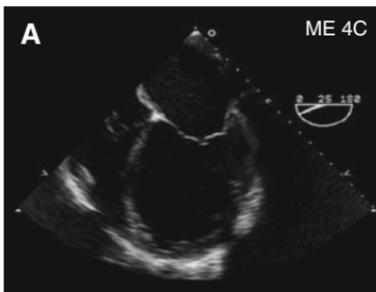
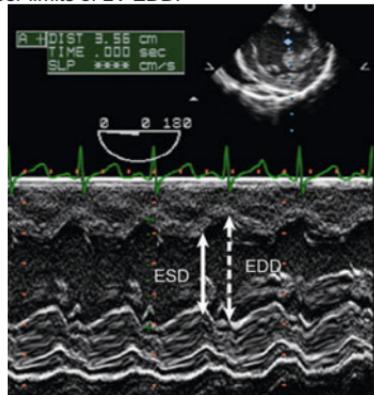
Mild (57–63 mm)  
 Moderate (61–68 mm)  
 Severe (> 62–69 mm)

### Causes of LV Dilatation

- CAD
- Valvular (MR, AI, AS)
- Cardiomyopathy (viral, idiopathic)
- Metabolic (Beriberi, thyrotoxicosis)
- Chagas disease
- Cocaine

### LV M-mode (TG SAX)

Used to measure end diastolic (EDD), end systolic (ESD) cavity dimensions, and wall thickness.



(A) ME 4C view shows dilated LV, at EDD, with poor MV leaflet coaptation during systole. With biventricular enlargement, the chambers will look proportional in size relative to one another, but enlarged by measurement. (B) TG SAX view shows thinned myocardium and spontaneous echo contrast ("smoke") in the LV.

## Ventricular Dimensions

### Right Ventricle Size

RV wall thickness < 5 mm EDD, use inferior or lateral walls as less epicardial fat  
RV hypertrophy (RVH) > 7 mm, occurs with pressure overload from:

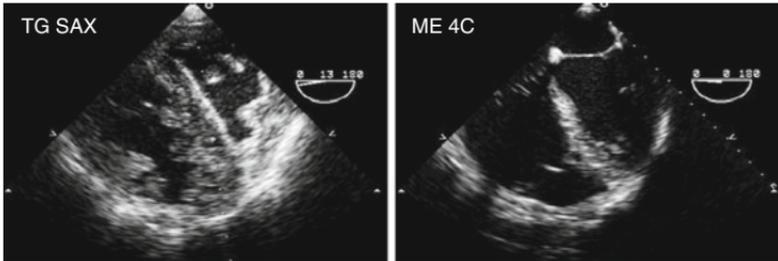
- Pulmonary hypertension
- RVOT obstruction (valvular, subvalvular, supra-valvular)
- Infiltrates (amyloid)

RV is normally triangular in shape, and the apex is formed by the LV (ME 4C view):

- RV area < 0.6 LV area
- RVED volume (49–101 mL/m<sup>2</sup>) is actually more than LVED volume
- RV length < 0.6 LV length

Dilated RV	Causes of RV Dilatation
Results from RV volume overload Apex formed by RV	<ul style="list-style-type: none"> <li>• CAD (MI)</li> <li>• Pulmonary hypertension</li> <li>• ASD/VSD</li> <li>• Pulmonic insufficiency/TR</li> <li>• Dilated cardiomyopathy</li> <li>• Congenital</li> </ul>
Mild: RV area 60% of LV area Moderate: RV area = LV area Severe: RV area > LV area	

TG view shows RVH, dilatation, and D-septum; 4C view shows apex formed by RV.



**Pericardial constraint** restricts interventricular septal (IVS) wall motion.

Findings associated with abnormal constraint:

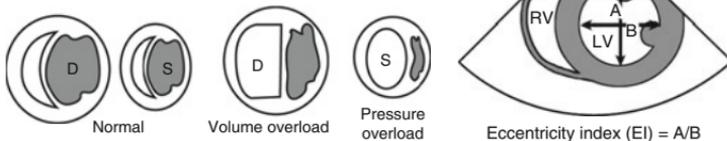
- Reciprocal ventilatory changes in RV and LV size
- Greater respiratory variation during inspiration and expiration in TV + MV inflow
  - Normal spontaneous: TV < 15%, MV < 10%
  - Constrictive pericarditis: TV > 40%, MV > 25%
  - Tamponade: TV > 85%, ↓ MV > 40% with inspiration

IVS is normally convex towards the RV during the entire cardiac cycle.

- RV pathology results in “D-shaped” septum with abnormal (paradoxical) motion, towards the LV during different parts of the cardiac cycle.

- Assess using **Eccentricity Index (EI)**

- Normal = 1 at EDD and ESD
- RV volume overload (EDD) EI > 1, ESD = 1
- RV pressure overload (ESD, EDD) EI > 1



## Ventricular Dimensions

**Table 1** Right ventricle and pulmonary artery size in DIASTOLE

	Reference range	Mild abnormal	Moderate abnormal	Severe abnormal
RV dimensions	Figure 1			
Basal RV diameter (RVD 1), cm	2.0–2.8	2.9–3.3	3.4–3.8	≥ 3.9
Mid-RV diameter (RVD 2), cm	2.7–3.3	3.4–3.7	3.8–4.1	≥ 4.2
Base-to-apex length (RVD 3), cm	7.1–7.9	8.0–8.5	8.6–9.1	≥ 9.2
RVOT diameters	Figure 2			
Below aortic valve (RVOT 1), cm	2.5–2.9	3.0–3.2	3.3–3.5	≥ 3.6
Below pulmonic valve (RVOT 2), cm	1.7–2.3	2.4–2.7	2.8–3.1	≥ 3.2
PA diameter	Figure 2			
Above pulmonic valve (PA 1), cm	1.5–2.1	2.2–2.5	2.6–2.9	≥ 3.0

RV Right ventricular; RVOT right ventricular outflow tract; PA pulmonary artery;

**Table 2** Right ventricle size and function as measured in ME 4 chamber view

Figure 1	Reference range	Mild abnormal	Moderate abnormal	Severe abnormal
RV diastolic area, cm <sup>2</sup>	11–28	29–32	33–37	≥ 38
RV systolic area, cm <sup>2</sup>	7.5–16	17–19	20–22	≥ 23
RV fractional area change, %	32–60	25–31	18–24	≥ 17

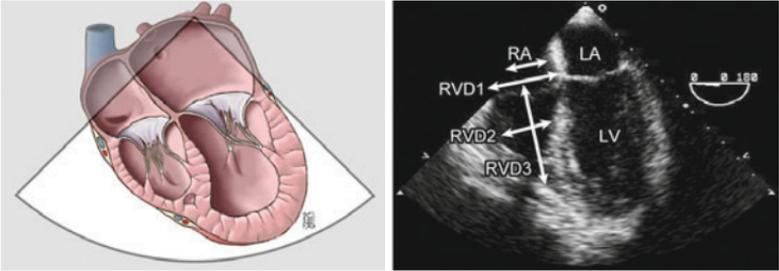
**Table 3** Left atrial and right atrial dimensions/volumes in SYSTOLE

Figure 1	Women				Men			
	Reference range	Mild abnormal	Moderate abnormal	Severe abnormal	Reference range	Mild abnormal	Moderate abnormal	Severe abnormal
Atrial dimensions								
LA diameter, cm	2.7–2.8	3.9–4.2	4.3–4.6	≥ 4.7	3.0–4.0	4.1–4.6	4.7–5.2	≥ 5.2
LA diameter/BSA, cm/m <sup>2</sup>	1.5–2.3	2.4–2.6	2.7–2.9	≥ 3.0	1.5–2.3	2.4–2.6	2.7–2.9	≥ 3.0
RA minor-axis, cm	2.9–4.5	4.6–4.9	5.0–5.4	≥ 5.5	2.9–4.5	4.6–4.9	5.0–5.4	≥ 5.5
RA minor-axis/BSA, cm/m <sup>2</sup>	1.7–2.5	2.6–2.8	2.9–3.1	≥ 3.2	1.7–2.5	2.6–2.8	2.9–3.1	≥ 3.2
Atrial area								
LA area, cm <sup>2</sup>	≤ 20	20–30	30–40	> 40	≤ 20	20–30	30–40	> 40
Atrial volumes								
LA volume, mL	22–52	53–62	63–72	≥ 73	18–58	59–68	69–78	≥ 79
LA volume/BSA, mL/m <sup>2.8</sup>	16–28	29–33	34–39	≥ 40	16–28	29–33	34–39	≥ 40

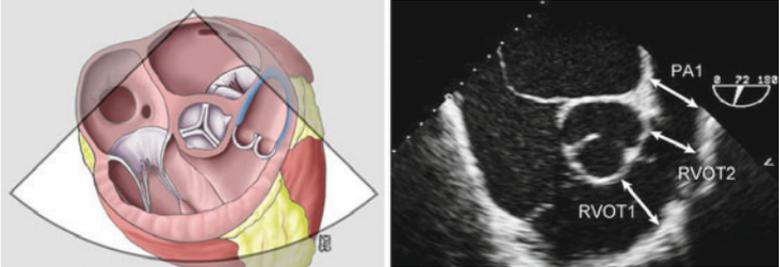
BSA body surface area; LA left atrial; RA right atrial. <sup>a</sup>Recommended and best validated

Adapted from: Lang RM, et al. *J Am Soc Echocardiogr* 2005;18:1440-63.

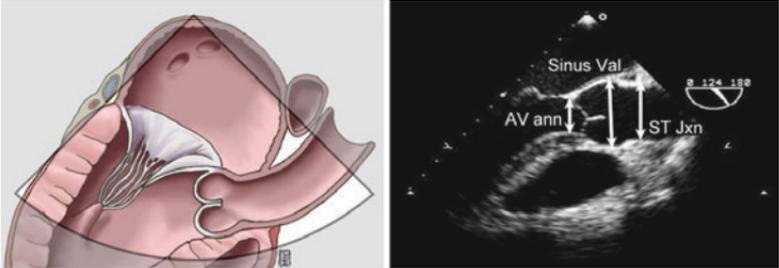
# Ventricular Dimensions



**Figure 1** TEE measurements of right ventricular (RV) diameters from ME 4 Chamber view, best imaged after optimizing maximum obtainable RV size by varying angles from approximately 0° to 20°.



**Figure 2** Measurement of right ventricular outflow tract (RVOT1) at pulmonic valve annulus (RVOT2) and main pulmonary artery (PA1) from ME RV Inflow–Outflow view.



**Figure 3** Measurement of aortic root diameters at aortic valve annulus (AV ann) level, sinuses of Valsalva (Sinus Val), and sinotubular junction (ST Jxn) from ME AV LAX view, usually at an angle of approximately 110°–150°. Annulus is measured by convention at base of aortic valve cusps. Although leading edge to leading edge technique is demonstrated for the Sinus Val and ST Jxn, some prefer inner edge to inner edge method.

## Ventricular Dimensions

**Table 4** Left ventricle size

Figure 4	Women				Men			
	Refer. range	Mild abnormal	Moderate abnormal	Severe abnormal	Refer. range	Mild abnormal	Moderate abnormal	Severe abnormal
<b>LV dimensions (DIASTOLE)</b>								
Diameter, cm	3.9–5.3	5.4–5.7	5.8–6.1	≥ 6.2	4.2–5.9	6.0–6.3	6.4–6.8	≥ 6.9
Diameter/BSA, cm/m <sup>2</sup>	2.4–3.2	3.3–3.4	3.5–3.7	≥ 3.8	2.2–3.1	3.2–3.4	3.5–3.6	≥ 3.7
Diameter/height, cm/m	2.5–3.2	3.3–3.4	3.5–3.6	≥ 3.7	2.4–3.3	3.4–3.5	3.6–3.7	≥ 3.8
<b>LV volume</b>								
Diastolic volume, mL	56–104	105–117	118–130	≥ 131	67–155	156–178	179–201	≥ 201
Diastolic volume/BSA, mL/m <sup>2a</sup>	35–75	76–86	87–96	≥ 97	35–75	76–86	87–96	≥ 97
Systolic volume, mL	19–49	50–59	60–69	≥ 70	22–58	59–70	71–82	≥ 83
Systolic volume/BSA, mL/m <sup>2a</sup>	12–30	31–36	37–42	≥ 43	12–30	31–36	37–42	≥ 43
<b>2D Method</b>								
Ejection fraction, % <sup>a</sup>	≥ 55	45–54	30–44	< 30	≥ 55	45–54	30–44	< 30

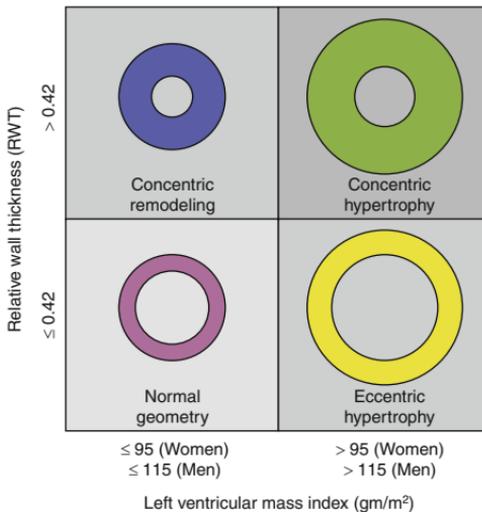
**BSA**, body surface area; **LV** left ventricle

<sup>a</sup>Recommended and best validated

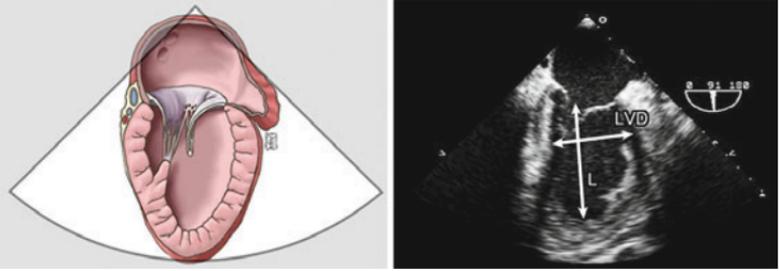
**Table 5** Left ventricle mass calculations (Figures 4–6)

LV Mass	=	$0.8 \times \{1.04[(LVIDd + PWTd + SWTd)^3 - (LVIDd)^3]\} + 0.6 \text{ g}$
RWT	=	$(2 \times PWTd) / LVIDd$

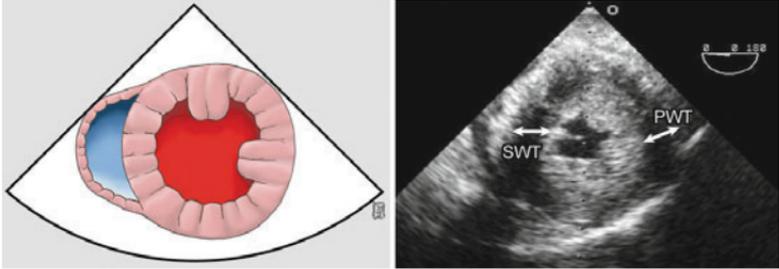
Measured at end diastole



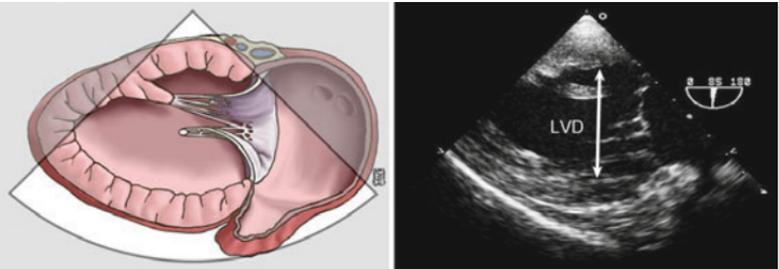
## Ventricular Dimensions



**Figure 4** TEE measurements during diastole of left ventricular length (L) and minor diameter (LVD) from ME 2 Chamber view, usually best imaged at an angle of approximately  $60^{\circ}$ – $90^{\circ}$ .



**Figure 5** TEE measurements of wall thickness of left ventricular (LV) septal wall (SWT) and posterior wall (PWT) from TG mid SAX view of LV, at papillary muscle level, usually best imaged at angle of approximately  $0^{\circ}$ – $30^{\circ}$ . Measure wall thickness at end diastole, exclude papillary muscles.



**Figure 6** TEE measurements of left ventricular (LV) minor-axis diameter (LVD) from TG 2 Chamber view of LV, usually best imaged at an angle of approximately  $90^{\circ}$ – $110^{\circ}$  after optimizing maximum obtainable LV size by adjustment of medial-lateral rotation.

## Ventricular Wall Motion

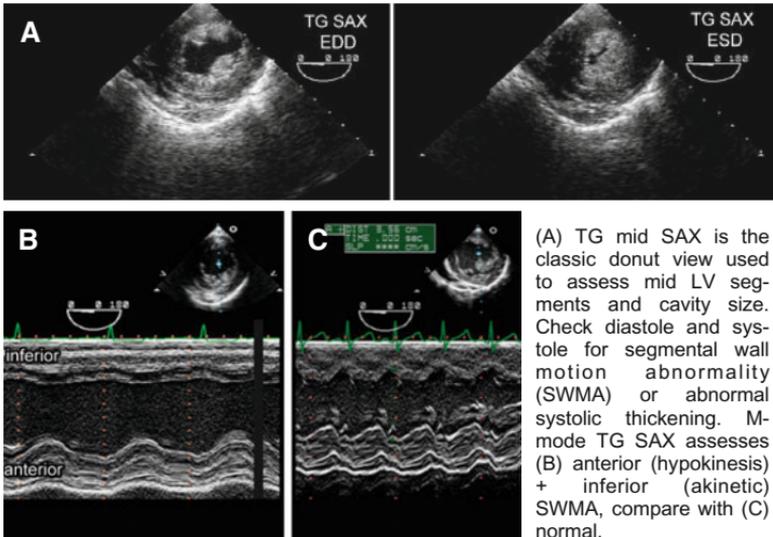
### Ventricle Function (Semiquantitative)

Assess ventricular function by examining systolic wall motion and systolic wall thickening. Tethering of adjacent wall segments can overestimate ischemic regions, so look closely at systolic wall thickening.

Wall score		Wall motion	% radius change	Wall thickening	
1	Normal	Inward	>30 %	+++	30–50%
2	Mild	Inward	10–30%	++	30–50%
3	Severe	Inward	< 10%	+	< 30%
4	Akinesis	None	None	0	< 10%
5	Dyskinesis	Outward	None	0	None

Wall motion score index (WMSI) =  $\frac{\text{Sum of wall motion scores}}{\text{Number of visualized segments}}$   
 Normal WMSI = 1, WMSI > 1.7 indicates perfusion defect > 20%

LV Dysfunction	RV Dysfunction
<ul style="list-style-type: none"> <li>• LV + LA enlarged</li> <li>• Segmental thinning or scarring</li> <li>• Reduced FAC<sup>a</sup></li> <li>• Decreased descent of base</li> <li>• Reduced MV and AV VTI's</li> <li>• ± regurgitant valves MV/TV</li> </ul>	<ul style="list-style-type: none"> <li>• Cavity enlarged RV &gt; LV</li> <li>• Crescent to oval shape</li> <li>• RV apex &gt; LV apex</li> <li>• Reduced free wall motion</li> <li>• <sup>b</sup>Septal flattening, dyskinesis</li> <li>• Quantification clinically impractical</li> </ul>
<sup>a</sup> FAC = fractional area change, VTI = velocity time integral <sup>b</sup> RV mass = LV mass ⇒ septal flattening RV mass > LV mass ⇒ paradoxical septal motion	



## Stroke Volume

**Stroke volume (SV):** Total amount of blood leaving the ventricle during systole, includes both antegrade + retrograde flow

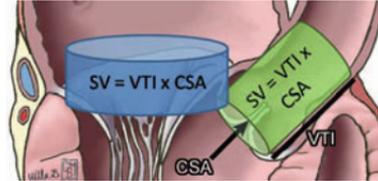
**Cardiac output (CO):** Amount of blood through the systemic circulation over time (L/min)

$$\text{Cardiac output (CO)} = \text{Stroke volume (SV)} \times \text{Heart rate (HR)}$$

Doppler estimate of stroke volume (SV):  
 CSA = cross sectional area =  $\pi r^2 = 0.785d^2$   
 VTI = velocity time integral is the cumulative distance (stroke distance in cm) that rbc's have traveled. Obtain by tracing the Doppler profile.

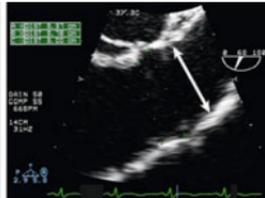
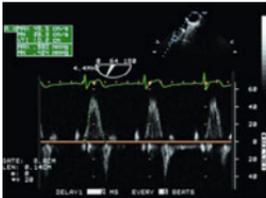
$$\text{SV} = \text{CSA} \times \text{VTI}$$

$$(\text{cm}^3) = (\text{cm}^2) \times (\text{cm})$$



### Right Ventricle Stroke Volume

VTI = pulmonary artery PW/CW X CSA =  $0.785d^2$  (d=PA or PV) = SV<sub>RV</sub>



$$\text{VTI} = 10.8 \text{ cm}$$

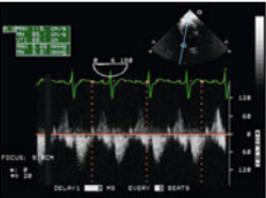
$$\text{CSA} = 0.785 (3 \text{ cm})^2$$

$$\text{SV} = 10.8 \times 9$$

$$= 97 \text{ cc}$$

### Transaortic Stroke Volume

VTI = aortic valve CW X CSA =  $0.785d^2$  (d= AV LAX) = SV<sub>transaortic</sub>



$$\text{VTI} = 22.1 \text{ cm}$$

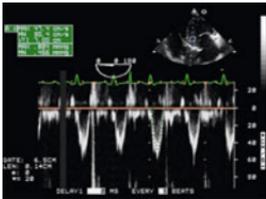
$$\text{CSA} = 0.785 (2.3 \text{ cm})^2$$

$$\text{SV} = 22.1 \times 4.15$$

$$= 92 \text{ cc}$$

### LVOT Stroke Volume

VTI = PW at LVOT X CSA =  $0.785d^2$  (d= TG LVOT) = SV<sub>LVOT</sub>



$$\text{VTI} = 16.8 \text{ cm}$$

$$\text{CSA} = 0.785 (2 \text{ cm})^2$$

$$\text{SV} = 16.8 \times 3.14$$

$$= 53 \text{ cc}$$

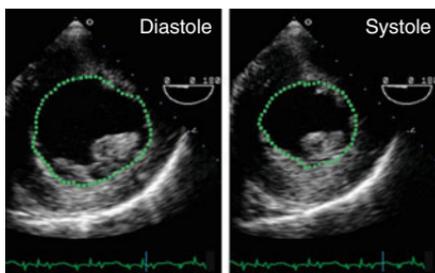
## Fractional Area Change and Shortening

### Global Systolic Function

Quantitative assessment of LV systolic function includes estimates of stroke volume (SV) and ejection fraction (EF). It is difficult to make volume calculations using TEE as assumptions are made about chamber shape and uniform global function. In addition, load conditions may affect indices of systolic function. Technical limitations include inadequate endocardial border definition and LV foreshortening.

#### Fractional Area Change (FAC)

LV systolic function using area measurements. This is not the ejection fraction as it is not a volume measurement. In TG mid SAX view planimeter, the endocardial area, excluding papillary muscles, using "eyeball" technique for largest (EDD) and smallest (ESD) size. Assumes normal global function with no SWMA.



$$\text{FAC (\%)} = \frac{\text{LVD area} - \text{LVS area}}{\text{LVD area}} \times 100\%$$

FAC 45 – 80% is normal

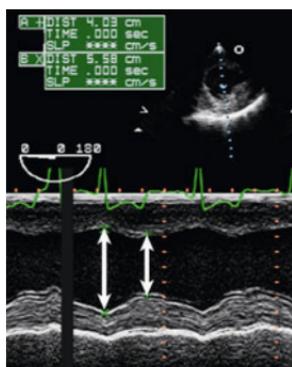
FAC < 20%: LV failure  
 FAC > 80%: hypovolemia or low SVR

#### Fractional Shortening (FS)

LV systolic function using linear measurements of end diastolic (LVIDd) and end systolic (LVIDs) internal diameters. FS only assesses mid or basal segments and poorly reflects overall LV function.

$$\text{FS (\%)} = \frac{\text{LVID}_d - \text{LVID}_s}{\text{LVID}_d} \times 100\%$$

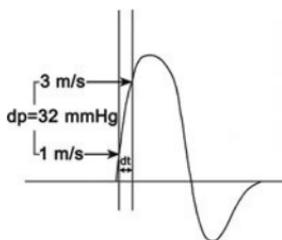
FS > 30% is normal



#### DP/dt

The rate of rise of intraventricular pressure (dp/dt) during systole can be estimated from the mitral regurgitation (MR) CW Doppler trace. This is an index of LV systolic function and may be less load dependent than FAC or EF. Measure the time (dt) taken for velocity to increase from 1 m/s to 3 m/s. The dp is calculated as 36 mmHg (3 m/s) – 4 mmHg (1 m/s) = 32 mmHg. Divide the dp/dt to obtain a value:

Normal > 1200 mmHg/s (dt < 26 ms)  
 Ventricular dysfunction < 800 mmHg/s (dt > 40 ms)



- Useful for patients with significant MR to assess LV function
- Not useful if trivial MR, eccentric jet, SWMA

# Ejection Fraction

## Ejection Fraction (EF)

Percent of LV diastolic volume ejected during systole can be obtained by calculating end systolic volume (ESV) and dividing by end diastolic volume (EDV). The stroke volume (SV) is the EDV – ESV. Ventricular volume can be calculated using the following methods.

$$EF \% = \frac{EDV - ESV}{EDV} \times 100\%$$

Normal EF = 55–75%

### Area–Length

- Trace endocardial border during ES and ED using a single view (ME 4C or ME 2C)
- Start and end at MV annulus which automatically closes the area loop
- Determine the LV apex
- Calculate

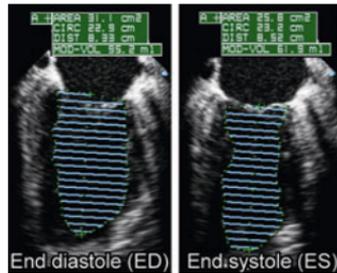
$$SV = \frac{0.85 (\text{area LV SAX})^2}{LV \text{ LAX length}}$$

- Normal SV = EDV – ESV:  
ME 4C = 57 ± 13 (37–94) cc/m<sup>2</sup>  
ME 2C = 63 ± 13 (37–101) cc/m<sup>2</sup>



### Method of Discs or Modified Simpson's Method

- Ventricle divided into discs
- Trace endocardial border during ED, ES in ME 4C and 2C views
- Begin and end at MV annulus
- Automatically calculates volume of 20 discs and sums for EDV and ESV
- Normal SV = EDV - ESV:  
55 ± 10 (36–82) cc/m<sup>2</sup>
- EF(%) = SV / EDV

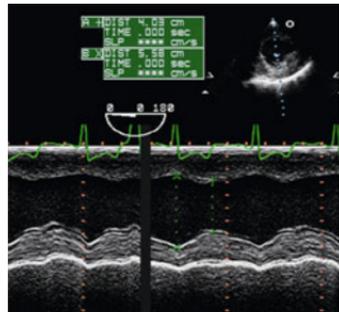


### Quinones

- TG mid SAX view
- M mode through the center, measure EDD, ESD diameters

$$EF \% = \frac{EDD^2 - ESD^2}{EDD^2}$$

- Assumes: normal global function  
normal LV shape
- Limitations: endocardial definition requires multiple measures, may multiply errors

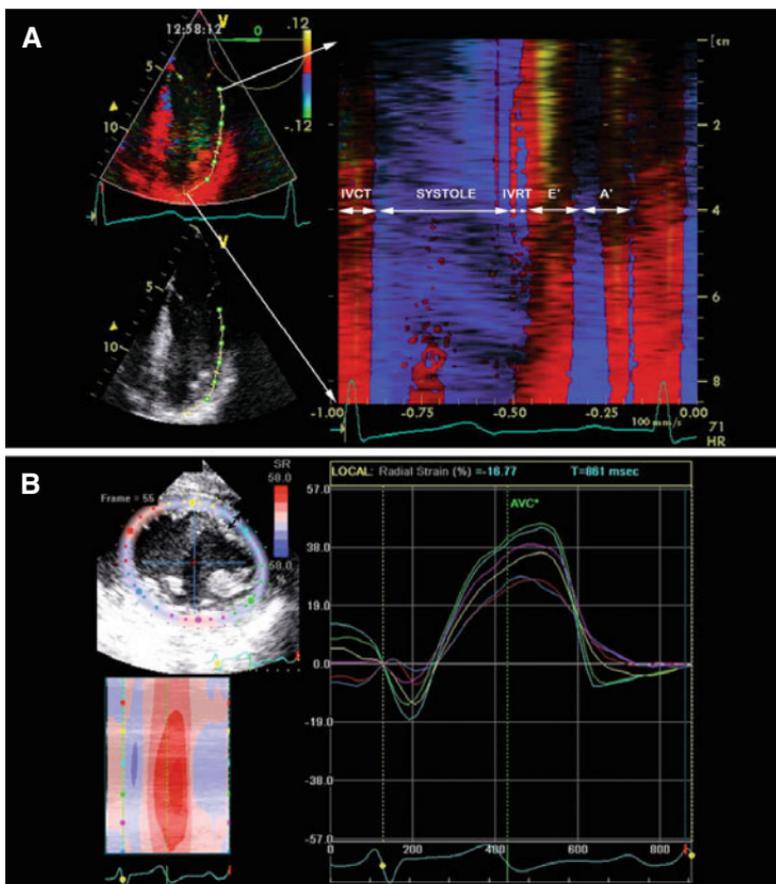


## Ventricular Strain

Myocardial strain/strain rate is the deformation of cardiac muscle and is an index for quantification of myocardial systolic function. It is calculated as percent or fractional change in dimension. The strain measure is named Langrangian strain.

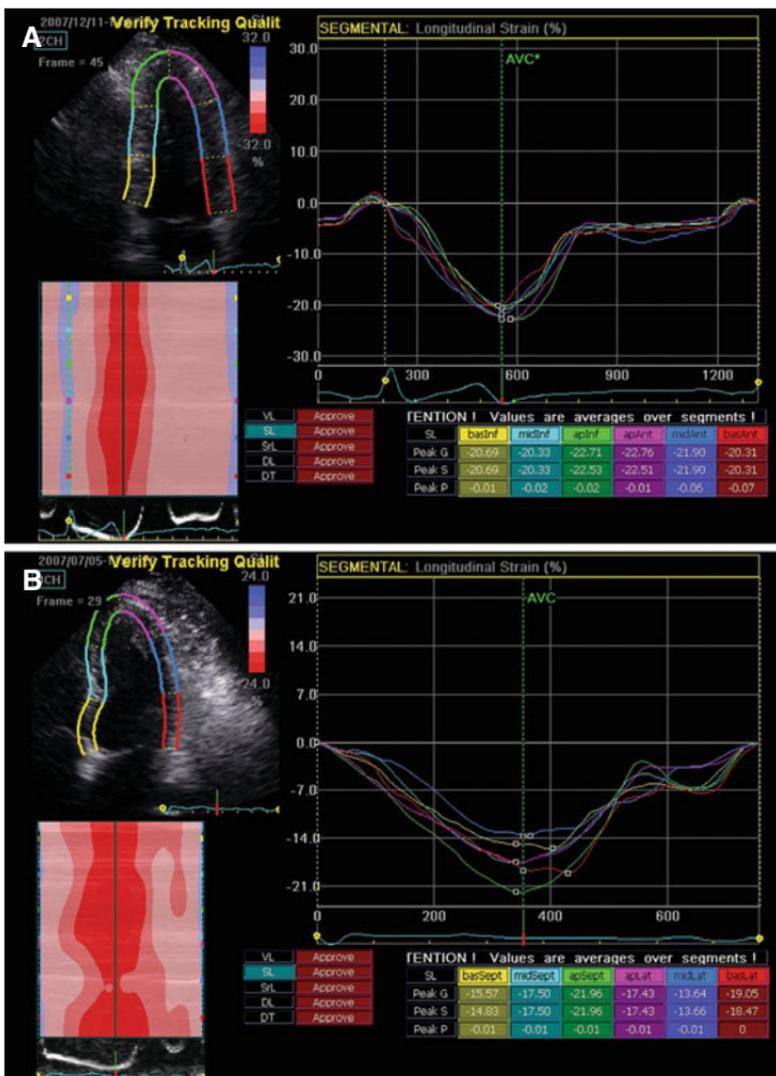
$$\text{Strain } (\epsilon) = (L - L_0) / L$$

Three types of strain can be measured: circumferential, radial strain (B TG short axis view), and longitudinal strain (from 4C, 3C, and 2C). By convention, lengthening and thickening are given positive values, whereas shortening and thinning are assigned negative values. Strain values can be obtained either from TDI or speckle-tracking imaging. The major advantage of speckle-tracking imaging is that it is independent of angle/cardiac translation. Tissue doppler Imaging (TDI) with curved M-Mode (A) of the lateral LV wall in the ME 4C view. Sample points were set on the lateral wall of the LV, and the velocities are plotted over time on the right.



## Speckle Tracking

Speckle-tracking imaging of the LV showing longitudinal strain in TTE. Normal strain pattern shows little deviation of each color coded segment (A) TTE speckle tracking 2C. Abnormal strain (B) TTE speckle tracking 2C shows as a wider deviation of each segment. The negative values suggest myocardial shortening.

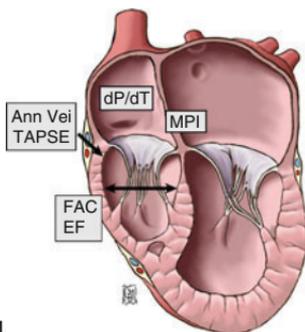


## Right Ventricular Function

### Right Ventricular Function

#### Indices of Systolic Function (normal values)

1. Geometric
  - RVEF (44–71%)
  - RVFAC (32–60%)
  - TAPSE (> 15 mm)
2. Myocardial velocity
  - Tissue Doppler annular velocity (> 12 cm/s)
  - Isovolumic acceleration ( $1.4 \pm 0.5$  m/s)
3. Hemodynamic
  - RV dP/dT
4. Time intervals
  - RV myocardial index (RVMPI) ( $0.28 \pm 0.04$ )



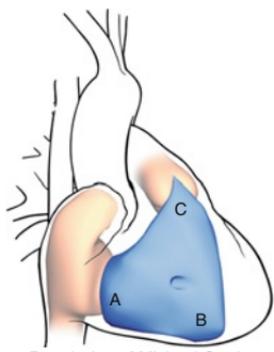
Source: Haddad F, et al. *Anesth Analg* 2009;108:407-21

Source: Rudski LG, et al. *J Am Soc Echocardiogr* 2010;7:685-713.

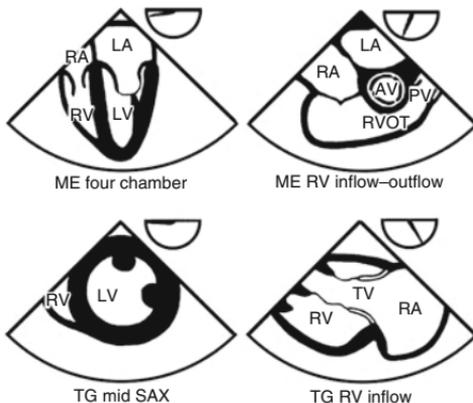
#### TEE Views to Assess RV Function

Anatomically RV is considered as three parts:

- A. Sinus (inflow) TV to apex, trabeculated myocardium
  - B. Apex free wall
  - C. Conus (outflow) to PV, smooth myocardium
- Moderator band
  - Papillary muscles

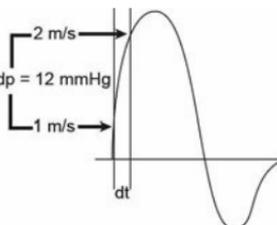


Permission of Michael Corrin



#### Right Ventricular dP/dt

- Index of RV contractility
- CW of tricuspid regurgitation (TR) trace
- $\Delta$  Pressure between 1 m/s and 2 m/s = 12 mmHg
- $dP/dt = \frac{12 \text{ mmHg}}{\text{Time for Velocity change 1 to 2 m/s in ms}}$
- Abnormal <400 mmHg/s
- Affected by loading conditions
- Can be used to trend if stable loading conditions



## Right Ventricular Function

### Right Ventricular Ejection Fraction (RVEF)

$$\text{RVEF} = (\text{RVEDV} - \text{RVESV}) / \text{RVEDV}$$

- Load dependent, prognostic value
- Use Simpson's rule or area length method
- Normal value 45–68%

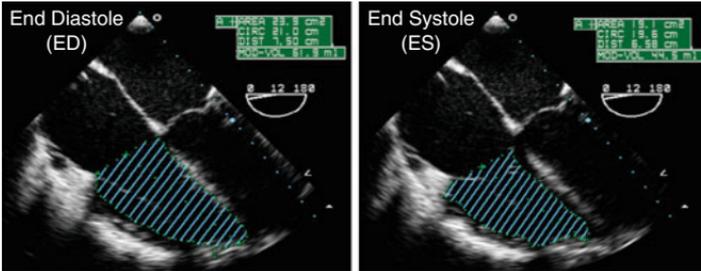
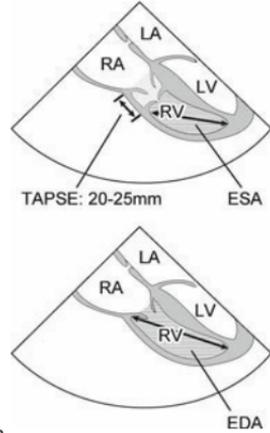
### Right Ventricular Fractional Area Change (RVFAC)

$$\text{RVFAC} = (\text{RV EDA} - \text{RV ESA}) / \text{RV EDA}$$

- Trace RV areas in systole and diastole
- Correlates with RVEF if no regional dysfunction
- Normal 32–60%, mild 25–31%, mod 18–24%, severe <17%

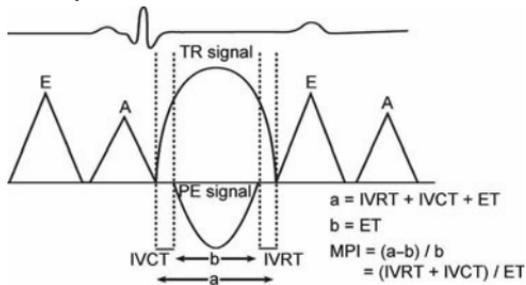
### Tricuspid Annular Plane Systolic Excursion (TAPSE)

- Longitudinal motion of lateral TV annulus towards the cardiac apex
- Measure shortening using Mmode in ME 4C view
- Normal 20–30 mm, impaired systolic motion < 16 mm



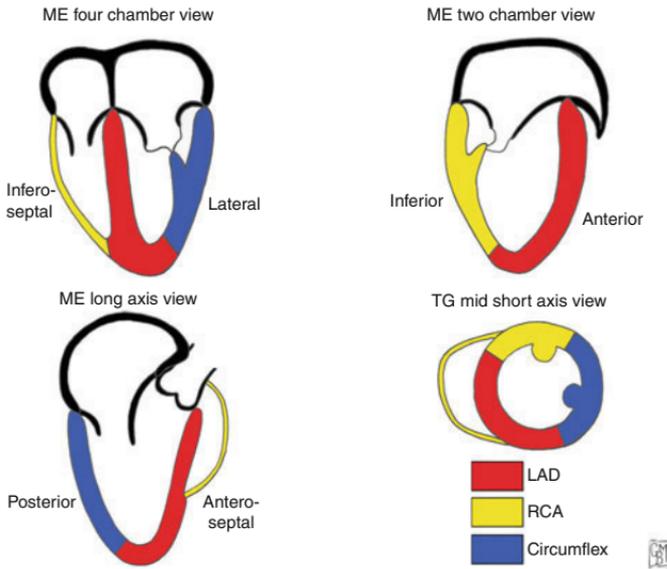
### Myocardial Performance Index (RVMPI)

- Obtain tricuspid regurgitation (TR) and pulmonic ejection Doppler traces
- $\text{MPI} = (\text{IVCT} + \text{IVRT}) / \text{ET}$
- Normal values: PW Doppler  $0.40 \pm 0.05$ , Tissue Doppler  $0.55 \pm 0.08$
- Increases in systolic and or diastolic dysfunction
- Less reliable with arrhythmias and 1° AV block



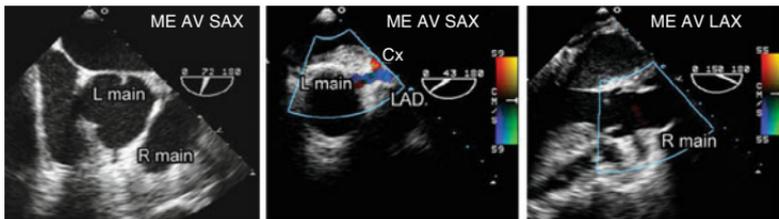
# Coronary Anatomy

## Coronary Anatomy



Left Coronary Circulation	Right Coronary Circulation
Left anterior descending (LAD) artery (anterior, antero/infero septal walls): <ul style="list-style-type: none"> <li>• Septal perforators</li> <li>• Diagonal branches</li> <li>• Posterior interventricular</li> </ul> Circumflex (Cx) artery (posterior, lateral walls): <ul style="list-style-type: none"> <li>• Obtuse marginal branches</li> <li>• Posterior interventricular</li> </ul>	Right coronary artery (RCA) (inferior wall, RV, AV + SA nodes): <ul style="list-style-type: none"> <li>• Posterior interventricular</li> <li>• Posterior lateral</li> <li>• Acute marginal</li> </ul> Papillary muscles blood supply: AL by two arteries (obtuse + diagonal) PM by one artery (RCA or obtuse)

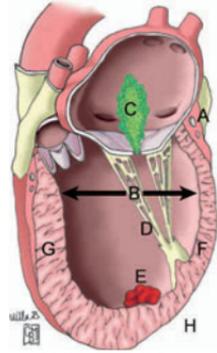
Dominance depends on which vessel (RCA, circumflex) supplies the posterior interventricular branch. The majority of hearts (85%) are right dominant.



## Ischemic Complications

### Ischemic Complications

- A. Chronic segmental dysfunction
- B. Ventricular dilatation
- C. Mitral regurgitation
- D. Papillary muscle dysfunction or rupture
- E. Thrombus
- F. Aneurysm
- G. Ventricular septal rupture
- H. Pericardial effusion



### Chronic Segmental Dysfunction

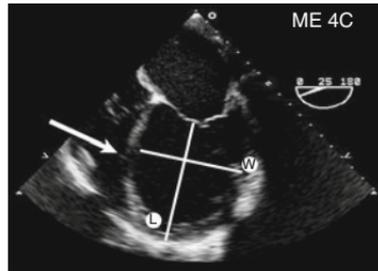
- Myocardial infarction (MI) is an irreversible injury to myocardium from prolonged ischemia. Myocardium is initially akinetic with normal wall thickness, over 4–6 weeks segments thin with increased echogenicity. Transmural MI has a definite area of akinesis and wall thinning. Nontransmural MI has hypokinesis and less wall thinning. Echocardiography cannot distinguish acute MI from ongoing ischemia.
- **Stunned myocardium:** Postreperfused viable myocardium with reversible post-ischemic dysfunction. Typically seen post-CPB.
- **Hibernating myocardium:** Segmental myocardial dysfunction due to impaired coronary perfusion. Can be assessed using Dobutamine Stress echocardiography. Shows a biphasic response to dobutamine, has initially improved wall motion with low dose that worsens with high dose dobutamine.

### LV Ventricular Dilatation

- Walls may be thinned (diastole)
- Bowed IVS (arrow)
- Smoke in the ventricle
- Displaced papillary muscles
- Measure EDD, ESD (TG views)

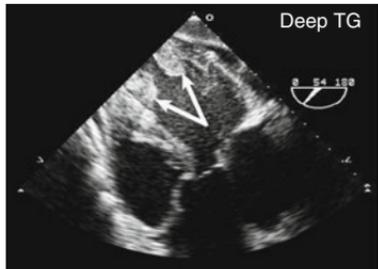
$$\text{Sphericity Index (EDD)} = \frac{\text{Length (L)}}{\text{Width (W)}}$$

Normal  $\geq 1.5$ ; severe:  $\leq 1$



### Thrombus

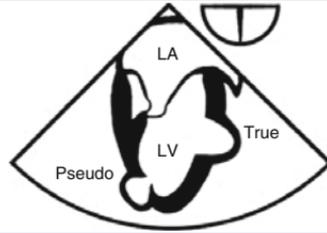
- 17–60% of MI patients
- More common anterior wall and apex
- Results from SWMA + blood stasis
- Locate SWMA (akinesis/dyskinesis)
- Echodense mass adjacent to SWMA
  - Early thrombus pedunculated
  - Late thrombus (>3 months) laminated
- Systemic embolization, up to 35%
- TTE gold standard to detect thrombus
- See in two TEE views: 2C, 4C, TG LAX
- Suspect if apex > IVS thickness



## Ischemic Complications

### Aneurysm and Pseudoaneurysm

- Transmural myocardial infarction (MI)
- Segmental wall motion abnormality (SWMA)
- Differentiate by size of orifice opening into LV and diameter of the aneurysm
- Color Doppler shows flow into and out of aneurysm
- Thrombus may be present



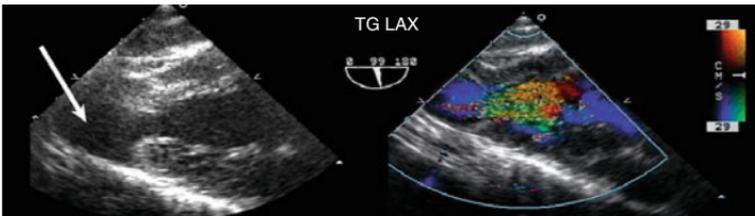
### Aneurysm

- 12–15% of MI patients, within 1<sup>st</sup> week, increased predictor of mortality
- Transmural infarct, commonly anterior wall + LV apex (LAD), infero-basal (Cx)
- TEE views (ME 2C, TG SAX, TG LAX)
- LV distorted wall in systole and diastole, noncontractile SWMA
- The walls of a true aneurysm always contain some myocardial cells
- Wide mouth, ratio of neck diameter: aneurysm diameter > 0.5
- Mural thrombi occur in 40–50%, low risk of rupture
- Chronic aneurysms persist > 6 weeks, are less compliant, and likely to expand during systole
- Patient may benefit from revascularization and restoration of ventricular geometry



### Pseudoaneurysm

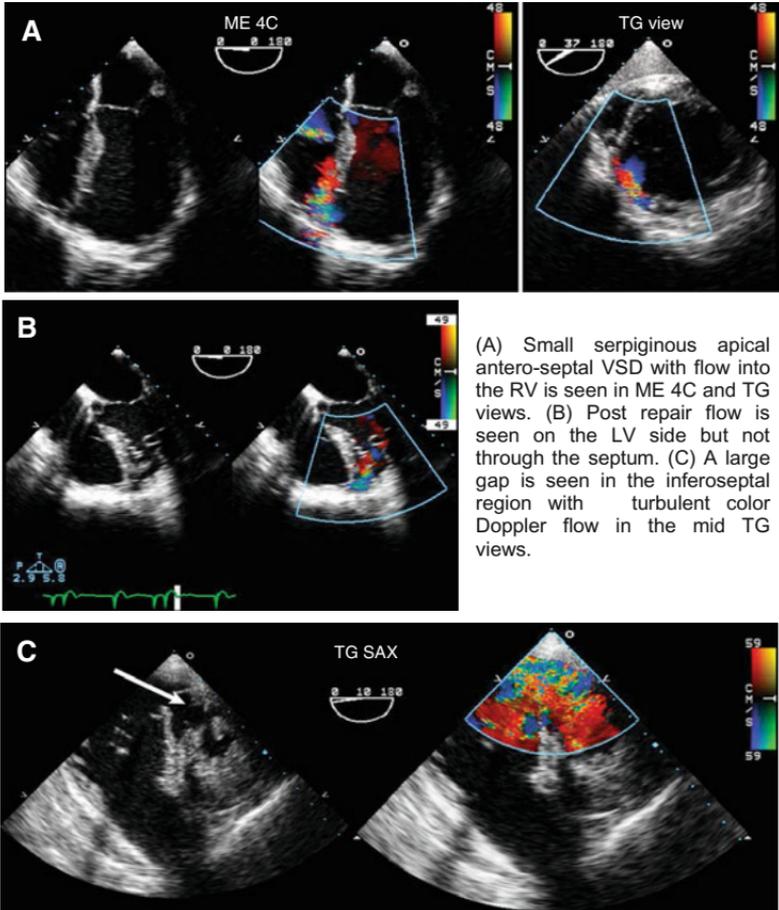
- Chronic contained ventricular rupture, wall composed of pericardium and clot
- Abrupt transition from normal myocardium to aneurysm, narrow neck at rupture site
- Ratio of neck diameter to maximum aneurysm diameter < 0.5
- In systole, pseudoaneurysm expands, flow in and out with color + PW Doppler
- Free rupture causes death
- May be partially filled with thrombus
- Life-threatening complication that requires immediate surgery to repair



## Ischemic Complications

### Ventricular Septal Defect (VSD)

- Rupture of interventricular septum (IVS) → communication between RV and LV
- 0.2% post-thrombolysis, 5% of deaths
- Occurs 2–5 days post-MI
- Most frequent antero-apical septum, also in posterior-basal septum (inferior MI)
- Thinned myocardium, SWMA, remainder of LV walls are hyperdynamic
- Simple direct hole or serpiginous, measure size (mm to cm)
- May be difficult to detect rupture site, use color Doppler to show turbulent flow
- CW Doppler: high velocity systolic L → R jet across IVS, estimate RVSP
- Calculate shunt fraction as pulmonary flow/aortic flow
- RV dysfunction, RV dilatation, paradoxical septal motion



## Ischemic Complications

### Ischemic Mitral Regurgitation (MR)

- Common, anterior MI (15%), inferior MI (40%)
- MR severity related to size of wall motion abnormality

Etiologies (based on MV leaflet mobility):

- Normal: annular dilatation ± perforation
- Excessive: prolapse ± flail ± torn chordae ± papillary muscle (PM) rupture
- Restricted: displaced PM ± LV dysfunction

Source: Agricola E, et al. Eur J Echocardiogr 2008;9:207-21.

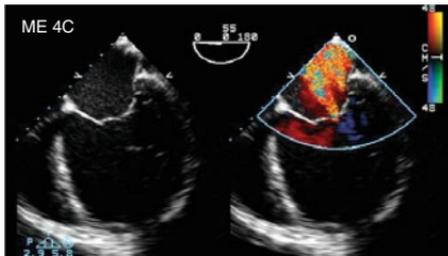
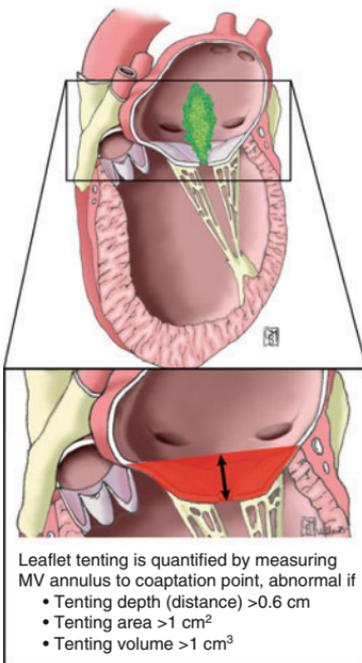
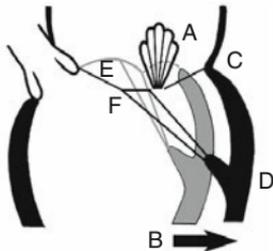
### Pathophysiology Ischemic MR

- Annular dilatation
- ↓ systolic transmitral pressure
- Apical + posterior displaced PMs
- Altered papillary-annular distance

↓  
Decreased leaflet mobility + malcoaptation

### TEE findings

- Central MR
- Dilated LV
- MV annulus dilated
- Posterior and apical displaced PM
- ↓ PM – annular angle
- Tethering MV leaflet (seagull)



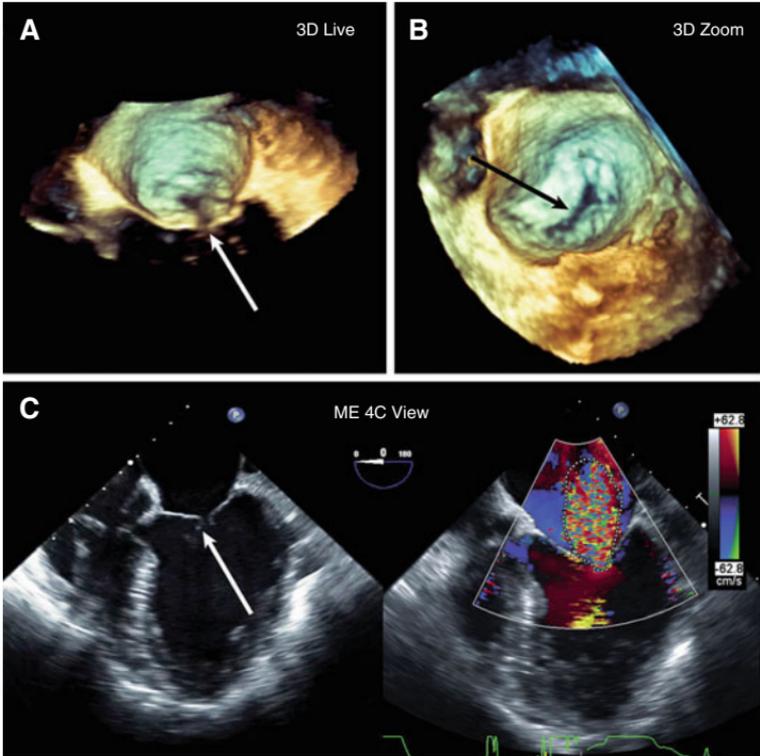
### Papillary Muscle Rupture

- 1% of MI, 2–7 days post-MI
- Commonly inferior MI, post-PM involved
- Partial PM rupture, mobile mass prolapses into LA
- Mitral regurgitation quantification
- ↑ mortality
- Surgical repair or MVR



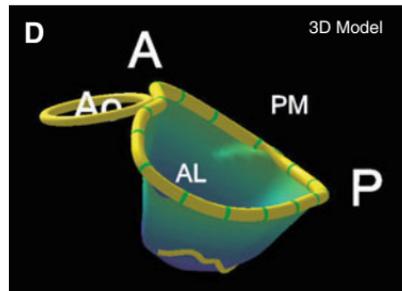
## Ischemic Complications

Patient with restricted MV leaflet motion from ischemic dilated cardiomyopathy shown during systole using real time 3D TEE from the LA side in (A) 3D Live and (B) 3D Zoom. Note the lack of central coaptation (arrows). (C) Compare 2D ME 4C view showing MV leaflet malcoaptation with severe central MR. (D) Reconstructed 3D model shows severe bileaflet restriction and tethering.



### Ischemic Mitral Valve

- Normal leaflet thickness
- Restricted leaflet mobility
- Central leaflet malcoaptation
- Central MR
- Annular dilatation
- Chordal tethering
- Leaflet tenting quantify need for MVR
  - Tenting area > 1 cm<sup>2</sup>
  - Tenting volume > 3.9 cm<sup>3</sup>



# Native Valves

Aortic Valve	
Anatomy .....	78
TEE Views .....	79–80
Bicuspid Aortic Valve.....	81
Aortic Stenosis.....	82–85
Aortic Insufficiency.....	86–89
Mitral Valve	
Anatomy .....	90–92
TEE Views .....	93–95
Mitral Regurgitation .....	96–103
Mitral Stenosis .....	104–107
Tricuspid Valve	
Anatomy .....	108
TEE Views .....	109
Tricuspid Regurgitation .....	110–111
Tricuspid Stenosis .....	112
Pulmonic Valve	
Anatomy and TEE Views .....	113
Pulmonic Insufficiency.....	114
Pulmonic Stenosis .....	115

## Aortic Valve Anatomy

### Normal Aortic Valve (AV) and Aortic Root Anatomy

The aortic valve is part of the aortic root that includes the aortic annulus, aortic valve cusps, sinuses of Valsalva, sinotubular junction (STJ), and proximal ascending aorta.

**Aortic Annulus:** Describes the basal attachment of the AV cusps at the aorto-ventricular junction. Cusps attach to 45% ventricular muscle (IVS) and 55% to fibrous tissue (AMVL). There is no fibrous or true ring-shaped anatomic AV annulus, instead have crown-shaped base at aortoventricular junction.

**Three valve cusps:** Right (most anterior), Non (near IAS), Left (near PA)

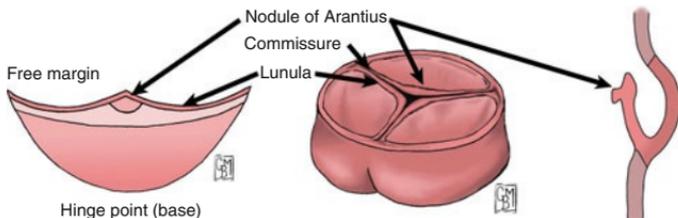
- Each cusp has a base, body, and free margin that attach in semilunar way.
- Hinge point is the basal point of cusp attachment at aortoventricular junction.
- Cusp free margin has a central thickened tip – the **nodule of Arantius** – and **lunulae** on either side, which serve as points of cusp coaptation.
- Lambl's excrescences are normal variant degenerative strands on the ventricular side of cusps.

**Commissures:** Area where two adjacent cusp margins meet the aorta.

**Interleaflet triangles:** Space between the base of two adjacent aortic valve cusps that are part of LVOT, exposed to ventricular pressures.

**Sinuses of Valsalva:** Space between cusp and aorta formed from 3 ball-shaped outpouches that give rise to the coronary ostia (R, L) and are important to AV function.

**Sinotubular junction:** Superior attachment of the AV cusps to a nonlinear area where the upper portions of the sinuses of Valsalva meet the aorta



### Aortic Root Relationships

Cusps:

Non: AMVL, membranous IVS  
 Right: Membranous IVS, anterior LV  
 Left: AMVL, anterior LV

Inter-leaflet Triangles:

Non/R: RA, RV, TV (septal leaflet)  
 R/L: Potential space aorta and PA  
 L/non: LA, AMVL

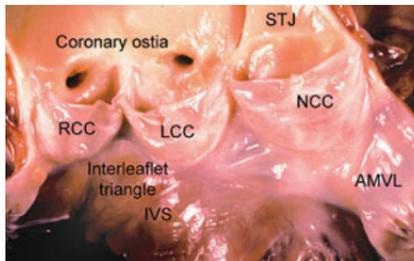
Coronary Sinuses of Valsalva:

Non: LA, RA, transverse pericardial  
 Right: RA, free pericardium  
 Left: LA, free pericardium

Source:

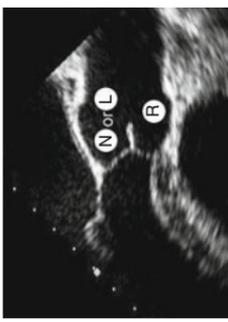
Ho S. Eur J Echocard 2009;10:i:3-10.

Free margin (FM) length = 28–34 mm  
 Cusp height = 13–16 mm  
 Cusp base = 42–59 mm (1.5 x FM length)  
 Cusp area: non > R > L  
 Hinge point: cusp attach to aorta

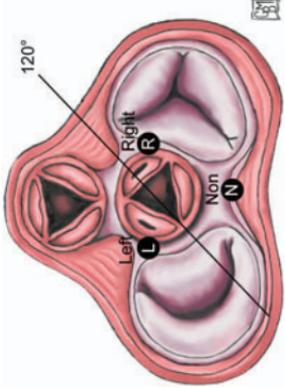


# TEE Views

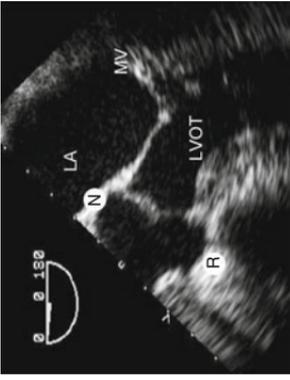
**ME AV LAX View (120°) in Diastole**  
 2D: RCC (ant), NCC or LCC (post) coaptation, prolapse  
 aortic root measurements  
 Color: AI direction (central, eccentric)  
 AS turbulent



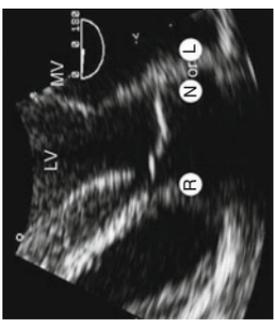
**Aortic Valve**  
 (oblique plane 30°–60°)



**ME 5 Chamber View (0°) in Diastole**  
 2D: RCC, NCC, LVOT, septal hypertrophy  
 Color: AI, systolic turbulence



**Deep TG View (0°) in Diastole**  
 2D: Prosthetic valve leaflet motion  
 Color: Paravalvular leak  
 CW: Doppler aligned for valve gradient



**TG LAX View (120°) in Systole**  
 2D: Cusp motion  
 Color: Paravalvular prosthetic valve leak  
 CW: Doppler aligned valve gradient



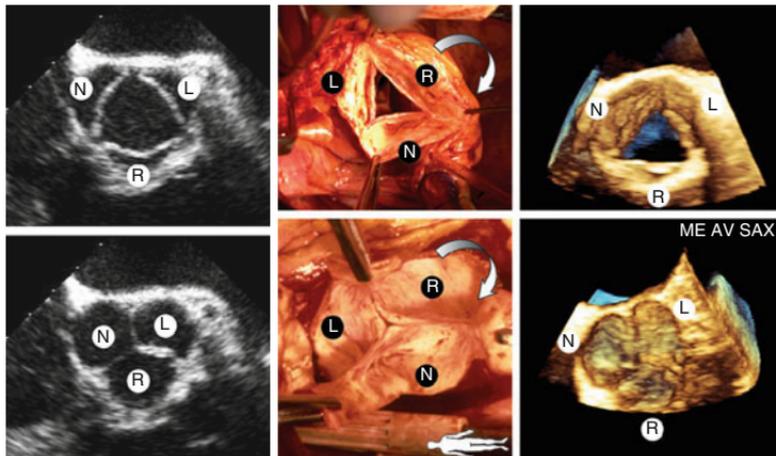
**ME AV SAX view (30°–60°) in Diastole**  
 2D: Number of cusps, coaptation, commissures  
 Color: AI location (central, commissural)  
 Advance (LVOT) and withdraw (coronary arteries) the probe to image



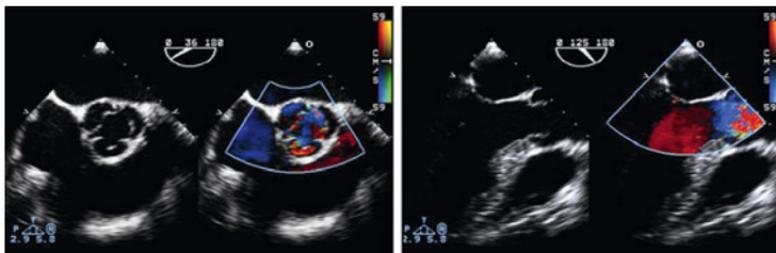
## TEE Views

### Aortic Valve ME SAX (30°) View and Surgeon's Perspective

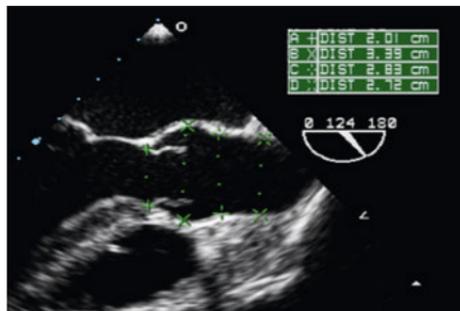
AV has a triangular opening during systole and closed during diastole forms Mercedes Benz sign. Rotate 90° for surgeon's view. Compare with 3D ME AV SAX views.



Normal systolic color Doppler flow in ME AV SAX (30°) and AV LAX (120°) views.



Measure Aortic Root dimensions during systole (largest) in ME AV LAX (120°) view.



- A. Annulus 18–25 mm
- B. Sinuses 24–39 mm
- C. STJ 22–29 mm
- D. Asc aorta 22–34 mm
- Root height<sup>a</sup> < 22 mm
- Root height/STJ < 0.8 mm

<sup>a</sup>root height is horizontal distance between the STJ and aortic annulus

# Bicuspid Aortic Valve

## Bicuspid Aortic Valve (BAV)

Two cusps:

Congenital: Equal or Unequal (usually anterior > posterior) cusp size

Acquired: Unequal cusp size from fused commissure

Describe commissure location (ie 4 + 10 o'clock), anterior/posterior, right/left

Raphe present at 90° to commissural opening

Thickened aortic cusps (may be mild)

Systolic elliptical orifice opening (SAX view)

Systolic cusp doming (LAX view)

Eccentric diastolic closure line (LAX view)

Diastolic doming from cusp prolapse (LAX view)

Usually three sinuses of Valsalva

Left ventricular hypertrophy (LVH)

Location of coronary ostia (usually 180° apart)

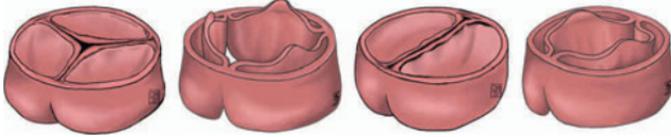
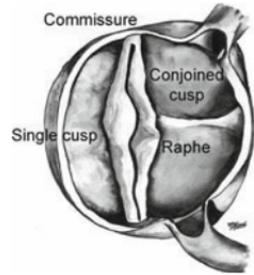
Type 1: Two coronary arteries anterior to valve orifice

Type 2: Valve orifice separates the coronaries

Associated pathology: AI, PDA, VSD

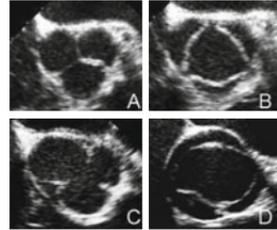
Aortopathy: dilatation, aneurysm, dissection

Coarctation of aorta in 15–20% BAV, 80–85% of coarctations have BAV



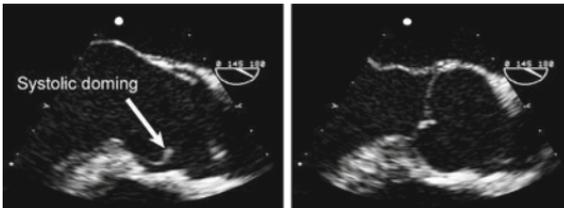
## Bicuspid Aortic Valve SAX

The number of AV cusps is determined during systole in the ME AV SAX view. In a normal AV with three cusps, the opened orifice is triangular (B). With a bicuspid valve, the orifice is oval or "fish mouthed" in appearance (D). During diastole, a bicuspid valve raphe (C) may make the "Mercedes Benz" sign similar to a normal AV (A).



## Bicuspid Aortic Valve LAX

Normal AV cusps open and close in the center of the sinuses of Valsalva. Bicuspid AV cusp opening is frequently eccentric, and the cusps appear domed during systole (arrow) from incomplete opening. The diastolic coaptation line may appear eccentric and the body of the cusp may prolapse (diastolic doming).



# Aortic Stenosis

## Aortic Stenosis

- Etiology: valvular, subvalvular, supra-annular
  - Valvular: degenerative/calcific, rheumatic, congenital bicuspid
  - Subvalvular: membrane, HOCM, SAM
- 2D findings:
  - Valvular calcium location, etiology (degenerative vs rheumatic)
  - Restricted cusp opening
  - SAX: Number of cusps (tri vs bicuspid), planimetry (difficult if calcified)
  - LAX: systolic doming (< 15 mm opening, angle < 90°), annulus size
  - Aortic sclerosis: thickened cusps without hemodynamic significance
- Doppler:
  - Color: turbulence at level of obstruction
  - PW: locate level of obstruction
  - CW: peak/mean velocity and P gradients varies with flow
    - underestimate (↓ LV function, MR, poor Doppler alignment, L→R shunt)
    - overestimate (high CO, AI)
    - If LVOT  $V_{peak} > 1.5$  m/s or  $AV V_{peak} < 3.0$  m/s then use modified Bernoulli:
 
$$\text{peak gradient} = 4(AV V_{peak})^2 - (LVOT V_{peak})^2$$
    - CW: continuity equation VTI (LVOT, AV) for AV area
- Stenosis severity (severe see below)
  - Peak velocity > 4.0 m/s
  - Mean/peak Pressure gradients 40–50 mmHg
  - AV area < 1.0 cm<sup>2</sup> (planimetry, continuity equation)
- LVH, small, postero-basal hypokinesis, poor LV underestimates AS
- Poststenotic aorta dilatation, functional MR, MAC

	Valve area (cm <sup>2</sup> )	Indexed valve area (cm <sup>2</sup> /m <sup>2</sup> )	Peak velocity (m/s)	Pressure peak (mmHg)	Gradient (mmHg) mean	
					AHA	ESC
Normal	3.0–4.0		1.4–2.2	8–20		
Mild	> 1.5	> 0.85	2.6–2.9	20–40	< 20	< 30
Mod	1.0–1.5	0.6–0.85	3.0–4.0	40–70	20–40	30–50
Severe	< 1.0	< 0.6	> 4.0	> 70	> 40	> 50

Adapted from: Baumgartner H, et al. J Am Soc Echocardiogr 2009;22:1-23.

### What to tell the surgeon

#### Pre-CPB:

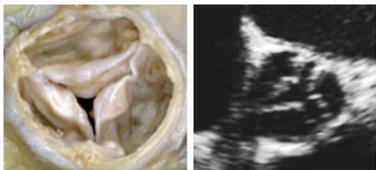
- Rheumatic, calcific etiology
- Annulus size (for stentless valve: STJ within 10%)
- Calcified AMVL (restricted motion) or aorta (difficult minimal access)
- AV area estimate (prosthetic mismatch), pressure gradients
- Poststenotic root dilatation, location of coronary ostia
- LVH (concentric), septal hypertrophy (SAM, LVOT diameter)

#### Post-CPB:

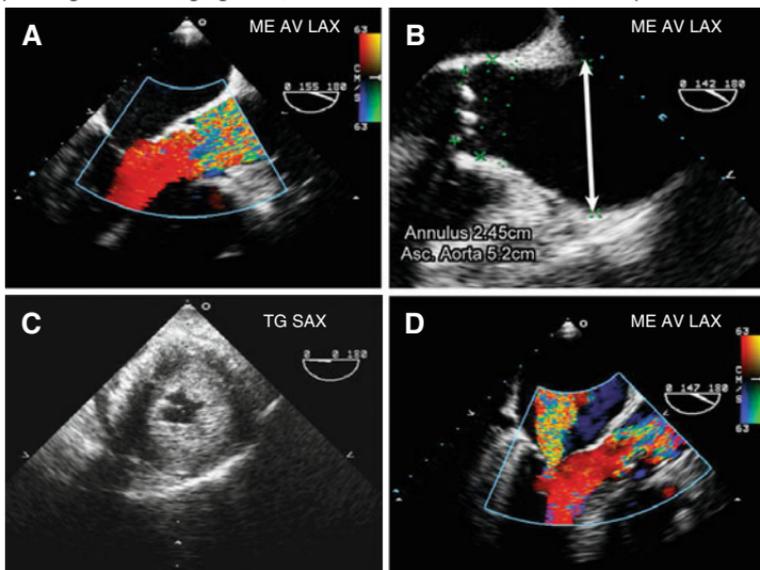
- Prosthetic valve stability, leaflet mobility
- Paravalvular, valvular leaks
- Peak/mean pressure gradients
- No LVOT obstruction with SAM or VSD (rare)
- Ventricular function (right and left), intra-cavitary gradient (↑ mortality)

## Aortic Stenosis

Aortic stenosis	2D findings	Color	Spectral Doppler
ME 5C (0°)	AV, subvalve	LVOT turbulence	Nonparallel
ME AV SAX (30°)	AV, planimetry	AV	
ME AV LAX (120°)	Sub/supra/AV	LVOT/AV/supra	
TG LAX (120°)	Sub/supra/AV	LVOT/AV/supra	CW (AV) PW (LVOT)
Deep TG (0°)	Difficult image	LVOT/AV/supra	

Calcified aortic valve	Rheumatic aortic valve
<p>Calcific, degenerative, thick stiff cusps, base of cusps calcified, stellate opening no commissural fusion Shadowing on 2D ME AV SAX view</p>	<p>Commissural fusion, triangular opening free borders calcified, calcific nodules on both surfaces, rheumatic MV Some calcium in 2D ME AV SAX view</p>
	

(A, B) ME AV LAX shows systolic doming, restricted opening, and turbulent flow at the AV into ascending aorta. (B) Post-stenotic ascending aorta dilatation may require aorta replacement. (C) LV hypertrophy results in small stroke volume, diastolic dysfunction, and inferior wall hypokinesis. (D) Need to differentiate functional or 1° pathological mitral regurgitation, as the latter will need additional MV repair.



# Aortic Stenosis

**Continuity Equation** for physiological (or effective) aortic valve area (AVA)

$$A_{AV} = \frac{VTI_{LVOT} \times 0.785 d^2_{LVOT}}{VTI_{AV}}$$

## AS Velocity

CW in TG views (deep TG, TG LAX):

- ↓ gain and ↑ wall filter
- Adjust to fill velocity axis

Dense velocity curve, identifiable peak:

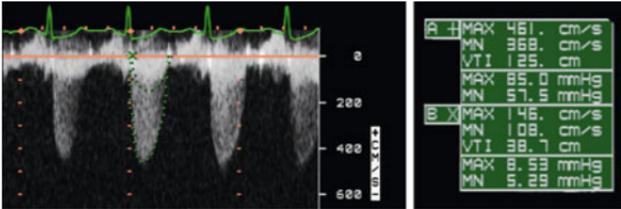
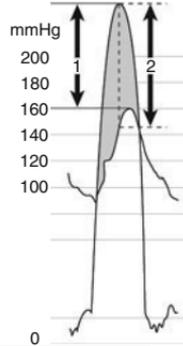
- Trace outer edge for VTI and mean Pressure gradient
- Peak maximum instantaneous Doppler is higher (2)
  - Peak to peak cath gradient is lower (1)
  - Mean doppler and cath gradients are similar

Shape distinguishes obstruction level:

- More severe = rounded + midsystole

Error:

- Misaligned Doppler
- Pressure recovery (PR) if ascending aorta < 30 mm



## LVOT Velocity

PW in TG views:

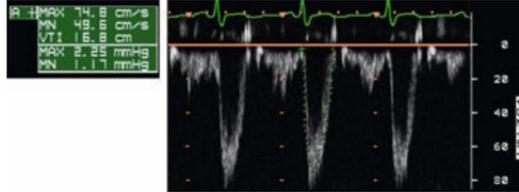
- Gate 3–5 mm
- Low wall filter

Smooth velocity curve:

- Well defined peak
- Narrow velocity range
- Identify peak velocity
- Trace modal velocity for VTI

Error:

- Improper position, spectral broadening (filled in) too close to AV (overestimate)



## LVOT Diameter

ME AV LAX zoom mode, symmetrical aortic root:

- Optimal fibrous tissue boundary
- Inner to inner edge
- Mid-systole
- Parallel to AV plane, within 0.5 – 1 cm of AV

Assumes a circular CSA

Source of error if inaccurate measurement

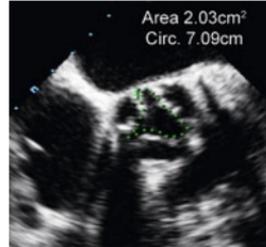


## Aortic Stenosis

Abnormally low gradient	Abnormally high gradient	AS velocity > 4 m/s + AVA > 1.0 cm <sup>2</sup>	AS velocity ≤ 4 m/s + AVA ≤ 1.0 cm <sup>2</sup>
LV dysfunction MR LVH (low SV)	AI High CO	High CO Mod–severe AI Large BSA	Low CO Severe MR Small BSA
Velocity and pressure gradients are flow dependent, ↑ flow will ↑ gradient and ↓ flow will ↓ gradient. Calculate AVA if gradients are unreliable			

### Planimetry

- ME AV SAX view
- Trace AV orifice during systole
- Obtain the anatomic AVA which differs from the effective AVA obtained by the continuity equation
- Limited if heavily calcified
- Accurate only at the smallest orifice



### Double envelope technique

Trace inner envelope (LVOT) and outer envelope (AV) for VTI and pressure gradients (peak and mean) use the continuity equation.

### Low velocity + Low gradient AS

Effective AVA < 1.0 cm<sup>2</sup>

LVEF < 40%

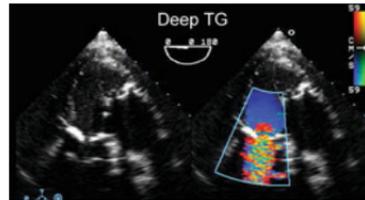
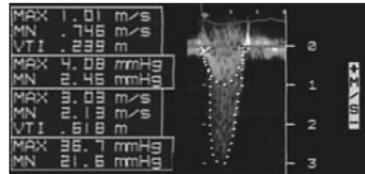
Mean PG < 30–40 mmHg

Consider Dobutamine stress test

### Velocity ratio (VR)

A  $VTI_{LVOT} / VTI_{AV}$  ratio < 0.25 indicates severe AS. It is a dimensionless number, independent of CO, used to assess AS severity with poor LV function.

$$VR = \frac{VTI_{LVOT}}{VTI_{AV}}$$



### Prognosis

Depends on clinical symptoms and not hemodynamic measurements

Effective AVA (continuity) is 1° predictor of clinical outcome

Rate of progression (per year):

- Increase 0.3 m/s peak velocity
- Increase 7 mmHg peak gradient
- Decrease AVA 0.1 cm<sup>2</sup>

### Indications for Surgery

- Symptomatic patients with severe AS
- Mod–severe AS + CABG
- Mod–severe AS + other valve or aorta
- Replace aorta > 45 mm or calcified

## Aortic Insufficiency

### Aortic Insufficiency

- Etiology of Insufficiency:
  - Valve: prolapse, calcified, bicuspid, rheumatic, endocarditis
  - Dilated annulus + root: Marfan's, aneurysm, HBP, aortitis
  - Loss of commissural support: trauma, dissection, VSD
- 2D findings:
  - AV: # cusps, coaptation (SAX, LAX), diastolic fluttering or lack of cusp closure, prolapse, calcified/fused, bicuspid
  - Root dimensions: LVOT, annulus, sinuses, STJ, aorta (systole)
- Doppler findings:
  - Color: diastolic turbulence in LVOT, jet direction (LAX): central or eccentric, jet location (SAX): central or commissural
  - Color: measure JH/LVOT (LAX), Jet /LVOT CSA (SAX), vena contracta
  - CW: density, diastolic decay measured as PHT or deceleration slope
  - CW:  $\uparrow$  LVOT velocity > 1.5m/s
  - PW/CW: diastolic flow reversal in arch/descending/abdominal aorta
  - Calculate ERO area, regurgitant fraction (RF), regurgitant volume (RV)
- LV dilated, function variable
- Associated findings (indirect effect on MV): premature MV closure, reverse doming AMVL, fluttering AMVL, presystolic (diastolic) MR, jet lesion AMVL
- Severe insufficiency based on the following findings (ASE):
  - Specific: central jet JH/LVOT > 65%, vena contracta > 6 mm
  - Supportive: PHT < 200 ms, abd. aorta holodiastolic reversal,  $\uparrow$  LV size
  - Quantitative: RV > 60cc, RF > 50%, EROA > 0.3 cm<sup>2</sup>

### Aortic Insufficiency Severity (ASE/ACC)

Method	Mild	Moderate	Severe
Jet / LVOT width <sup>a</sup>	< 25%	25–64%	≥ 65%
Jet /LVOT CSA <sup>a</sup> (%)	< 5	5–59	≥ 60
CW density	Faint	Dense	Dense
PHT (ms)	> 500	200–500	< 200
Descending aorta reversal	Early brief	Intermediate	Holodiastolic
Vena Contracta <sup>a</sup> (mm)	< 3	3–6	> 6
ERO area (cm <sup>2</sup> )	< 0.10	0.1–0.29	≥ 0.30
Regurgitant Volume (cc)	< 30	30–59	≥ 60
Regurgitant Fraction (%)	20–30	30–49	≥ 50

<sup>a</sup>Nyquist limit 50–60 cm/s

Adapted from: Zoghbi W et al. J Am Soc Echocardiogr 2003;16:777-802.

#### What to tell the surgeon

##### Pre-CPB:

- Root vs valve pathology, dimensions of aortic root
- Number, morphology and coaptation of cusps (prolapse), calcified cusps
- Location + direction AI (central, eccentric commissural), AI severity
- Pulmonary valve annulus size (within 10-15% of aortic annulus for Ross)
- ± PI, R/O fenestrations (for Ross procedure)

##### Post-CPB:

- Cusp coaptation above the annular plane (LAX)
- Location and severity of AI

# Aortic Insufficiency

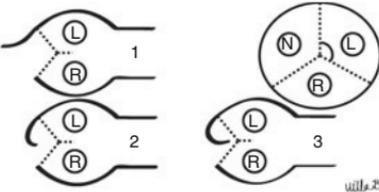
Views	2D findings	Color	Doppler CW
ME 5C (0°)	Root	Direction AI	Not aligned in ME views
ME AV SAX (30°)	Coaptation, # cusps	Location AI Severity AI	
ME AV LAX (120°)	Coaptation, root measure	Direction AI Jet length, width	
TG LVOT (120°)	Cusp motion	Paravalvular leak	Doppler aligned CW decay slope
Deep TG (0°)			

### AV Cusp Prolapse

Normal cusps coapt above the annular plane (dotted). Prolapse occurs if part of the cusp is below the annulus, three types:

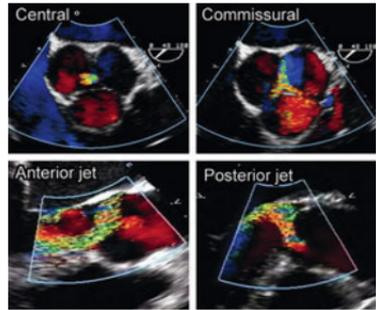
1. Flail: cusp tip in LVOT
2. Whole: free edge in LVOT
3. Partial: cusp body in LVOT

In LAX, see cusp below annular plane. In SAX, see double line or gap at cusp coaptation. Color shows AI location.



### AI Jet Direction/Location

In SAX, seen as continuous flow, locate diastolic flow as central or commissural, describe the cusp edges (R, L, non) involved. If eccentric AI jets seen in LAX (note direction as shown below), consider bicuspid AV, prolapsed or fenestrated cusp. Involved cusp is opposite to AI direction.

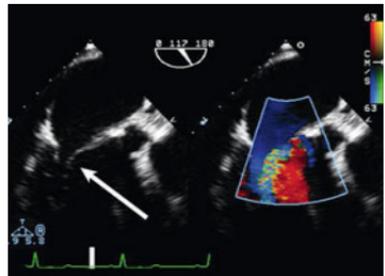
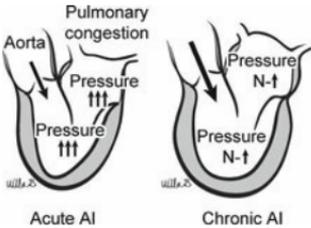


### Indirect Effects on Mitral Valve

- Premature MV closure
- Reverse doming of AMVL (arrow)
- Fluttering of IVS/AMVL
- Presystolic (diastolic) MR

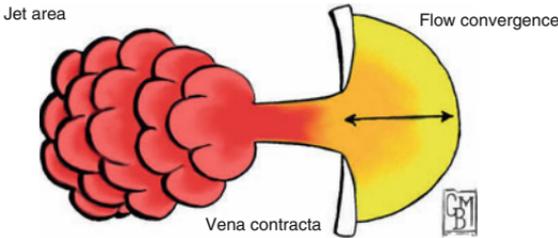
### LV Size and Function

- LV dilated
- Variable function



# Aortic Insufficiency

## Components of Aortic Insufficiency Jet



**Flow Convergence:** High velocity flow proximal to the regurgitant valve orifice results in a series of concentric hemispheres, termed flow acceleration. Adjust Nyquist limit ( $V_r$ ) to obtain a rounded flow convergence and measure the aliasing radius ( $r$ ). The PISA method can quantify AI severity by calculating the following:

**Effective Regurgitant Orifice area** (see pg. 47)

- Calculate AI flow across the valve:  $\text{Flow AI (cc/s)} = 6.28r^2 \times V_r \text{ (cm/s)}$
- Calculate EROA:  $\text{EROA}_{AI} \text{ (cm}^2\text{)} = \text{Flow AI (cc/s)} / V_{AI} \text{ (cm/s)}$

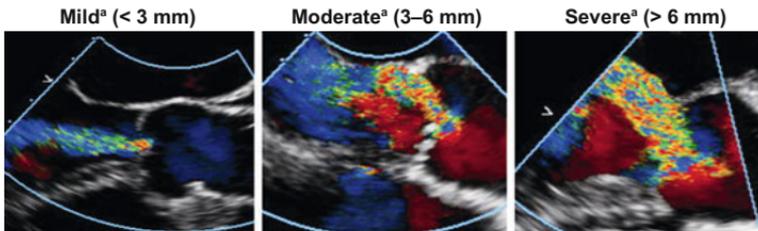
**Regurgitant Volume** (see pg. 48)

- Regurgitant volume (RV) is the volume through the effective regurgitant orifice:  
 $\text{RV (cc)} = \text{EROA}_{AI} \text{ (cm}^2\text{)} \times \text{VTI}_{AI} \text{ (cm)}$

**Regurgitant Fraction** (see pg. 48)

- Regurgitant fraction (RF) is percentage of regurgitant volume compared with total flow across the regurgitant valve ( $\text{RF} = \text{RV} - \text{SV}_{\text{normal}} / \text{RV}$ )

**Vena Contracta (VC)** is the narrowest portion of the jet at or just downstream from the orifice, with laminar flow and highest velocity. VC size is independent of flow rate and driving pressure for a fixed orifice, but may change with a dynamic orifice. VC is a sensitive semiquantitative measure of AI severity and can estimate the effective regurgitant orifice area ( $\text{EROA} = (\text{VC width}/2)^2$ , severe  $\geq 8\text{mm}^2$ ).



<sup>a</sup>Nyquist limit 50–60 cm/s

VC is difficult to accurately measure with TEE as seldom is the flow convergence region well seen due to color Doppler misalignment. VC should be measured below the region of flow convergence, between the AV cusps. Eccentric jets are measured perpendicular to the LAX. VC is not useful for assessing multiple jets.

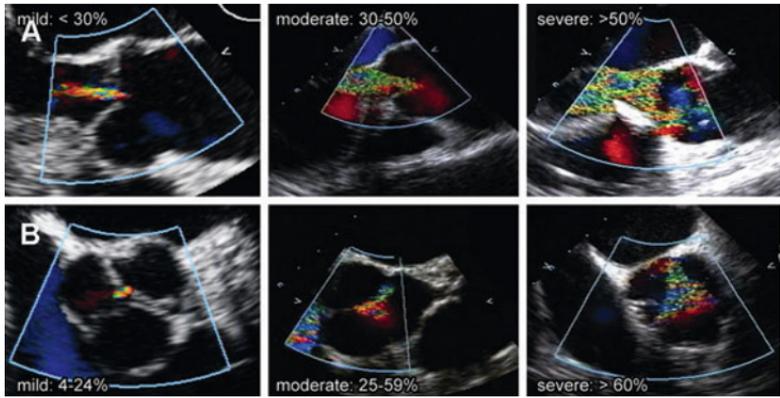
## Aortic Insufficiency

### Color Doppler

Assesses jet direction and AI severity by measuring jet area or height. Jet distance into the LV relies on hemodynamics and ultrasound machine settings, thus it is a poor measure of AI severity. Nyquist limit 50–60 cm/s.

(A) Jet height (JH) to LVOT height is measured in ME AV LAX view.

(B) Jet area to LVOT area measured below the AV cusps in the ME AV SAX view.



### Spectral Doppler Tracings

(A) CW trace: TG LAX or deep TG, assess density (compare with inflow), steepness, slope. Best alignment occurs if peak initial gradient  $> 40$  mmHg ( $V > 300$  cm/s).

(B) PW trace: Holodiastolic flow in the proximal arch (see below) and descending aorta is specific but not sensitive for severe AI. The more distal within the descending aorta, the greater the AI severity.

#### CW weak density

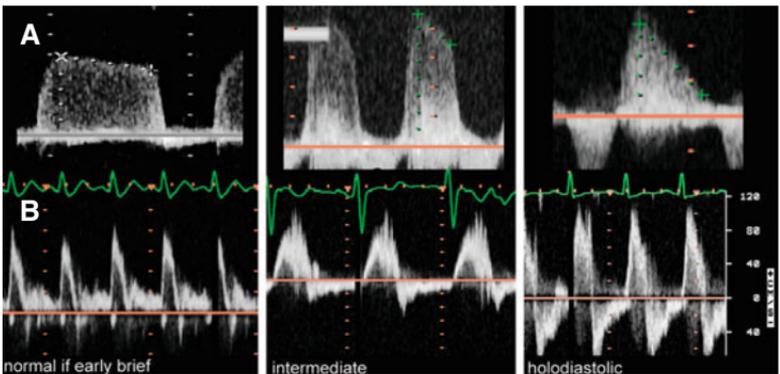
Mild – “flat top”  
Decay slope  $< 2$  m/s  
PHT  $> 500$  ms

#### CW moderate density

Moderate –  $\uparrow$  angle  
Decay slope  $2 - 3.5$  m/s  
PHT  $200 - 500$  ms

#### CW dense density

Severe – steep slope  
Decay slope  $> 3$  m/s  
PHT  $< 200$  ms



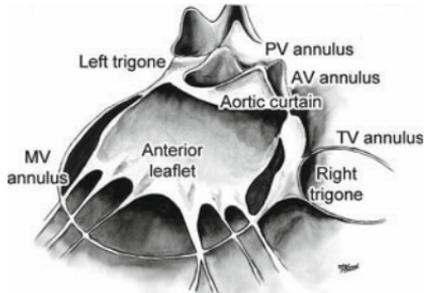
# Mitral Valve Anatomy

## Mitral Valve Anatomy

### Fibrous Skeleton (three parts)

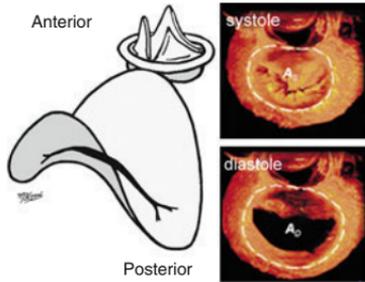
- Base aortic valve (AV)
- Right + left fibrous trigones  
intertriagonal distance (ITD)  
between R and L trigones  
 $ITD = AV \text{ diameter} / 0.8$
- Smaller fibrous area between  
RCC and pulmonary artery

"Aortic curtain" is fibrous and common to aortic + mitral valves.



### Mitral Annulus

- Posterior annulus has little fibrous tissue (P2 prolapse)
- Saddle-shaped (hyperbolic paraboloid) highest in ME 120° view
- Changes shape (small systole)  
Circle (diastole): 40% larger  
"D" shape (systole): smaller
- Measure annulus in diastole in Two views (0°, 90°)  
Normal size (29 ± 4 mm)
- Flexible annuloplasty ring changes shape

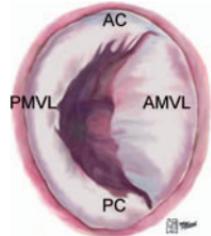


### Mitral Valve Leaflets

Four anatomic leaflets:

- Anterior (AMVL): 2/3 MV area, 1/3 MV annulus
- Posterior (PMVL): 2/3 MV annulus, 3 scallops
- Anterior commissure (AC)
- Posterior commissure (PC)

Leaflet nomenclature (see p. 92)  
Leaflet thickness ≤ 4 mm

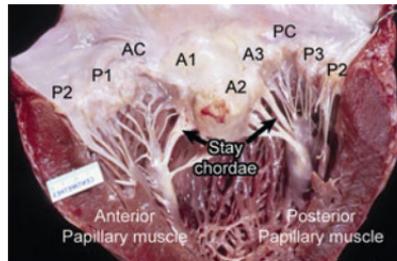


MV leaflet surface area is twice the annulus area (4–6 cm<sup>2</sup>) and allows for large leaflet coaptation area (30%).

### Chordae Tendineae

Three orders:

- 1<sup>st</sup>: leaflet free margin
  - 2<sup>nd</sup>: ventricular leaflet aspect
  - 3<sup>rd</sup>: vent wall to PMVL only
- stay chordae: attach to AMVL  
important for MV geometry



### Papillary Muscles (PM)

- Anterolateral: A2, A1, Ac, P1, P2
- Posteromedial: A2, A3, Pc, P3, P2
- Posterior PM has single artery supply (RCA or obtuse marginal of circumflex).

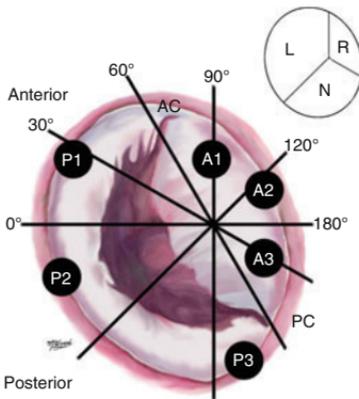
# Mitral Valve Anatomy

## Mitral Valve Orientation

### Anatomical

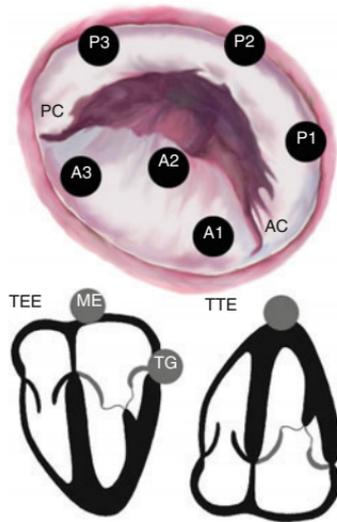
In TEE ME views the MV is imaged from above through the LA. The ME 4C view (0°) displays the AMVL on the left and PMVL on right. At > 90°, the PMVL appears on the left.

In TTE, the MV is imaged from below through the LV. The apical 4C view displays the AMVL on the left and the PMVL on the right. In TTE shadowing from the MV prevents adequate assessment of MR.



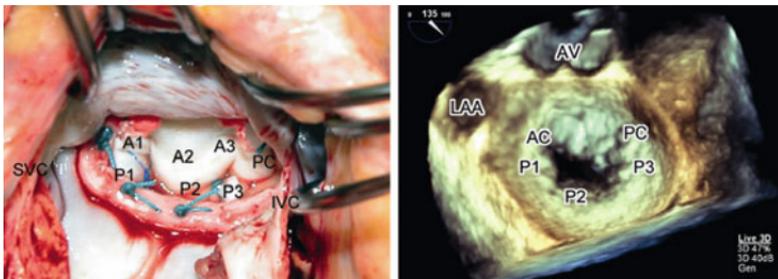
### TEE (TG SAX)

The MV is imaged from posterior, the LA is closest to the transducer. It is displayed with the AMVL in the far field and PMVL in near field (see pg. 14).



### Surgeon's View and 3D TEE

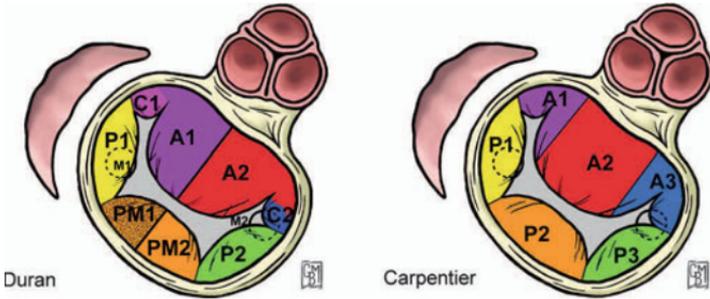
The MV as viewed by the surgeon through a left atriotomy is displaced 90° counter-clockwise from the 2D ME TEE views. The AMVL is superior and the PMVL is inferior, shown here with MV annuloplasty ring. The MV is imaged using real time 3D TEE can be orientated to display it in the surgeon's view as seen from the LA. In this image, the scallops of the PMVL are apparent. Note the aortic valve (AV) is displayed at the top of the image and the left atrial appendage (LAA) to the left.



# Mitral Valve Anatomy

Leaflet Nomenclature		
Anatomic	Duran	Carpentier
Posterior leaflet (scallops)		
Lateral	P1	P1
Middle	PM (1/2)	P2
Medial	P2	P3
Commissural leaflets		
Anterolateral	C1	Ac com
Posteromedial	C2	Pc com
Anterior leaflet (segments)		
	A1, A2	A1, A2, A3

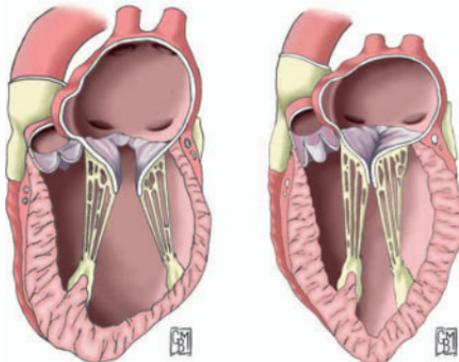
From Left Atrium, Anterior



## MV Function

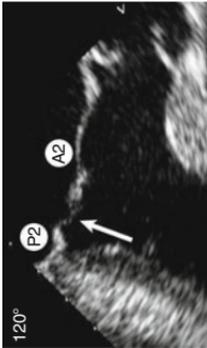
In diastole, the papillary muscles and LV myocardium relax, LA pressure exceeds LV pressure and the MV passively opens.

In systole, the papillary muscles contract making the chordae tendinae taut to prevent prolapse of the MV leaflets into the LA. The leaflet and chordae length are fixed. Excess leaflet area permits a large area of coaptation, analogous to a Roman arch.

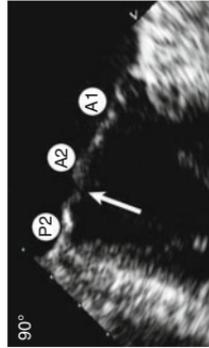
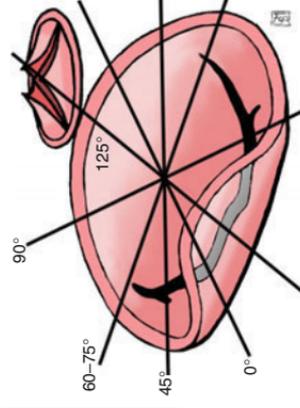


# TEE Views

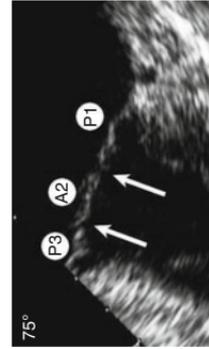
## TEE Midesophageal Views Mitral Valve



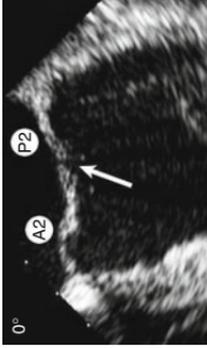
**ME AV LAX view (120°–135°)**  
AV in continuity with A2  
Annulus high point to detect prolapse  
Reliably image A2, P2



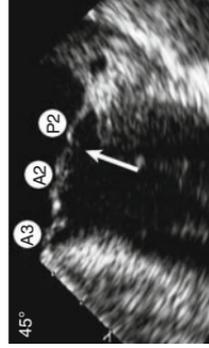
**ME 2C view (90°–105°)**  
AMVL is on the display right  
AMVL (A1-A2) is long and coaptis  
(arrow) with short P2



**ME Mitral Commissural view (60°–75°)**  
Image three distinct segments (P3, A2, P1)  
See two coaptation points (arrows)  
Mobile center A2 disappears in diastole (trapdoor)  
LAA is seen



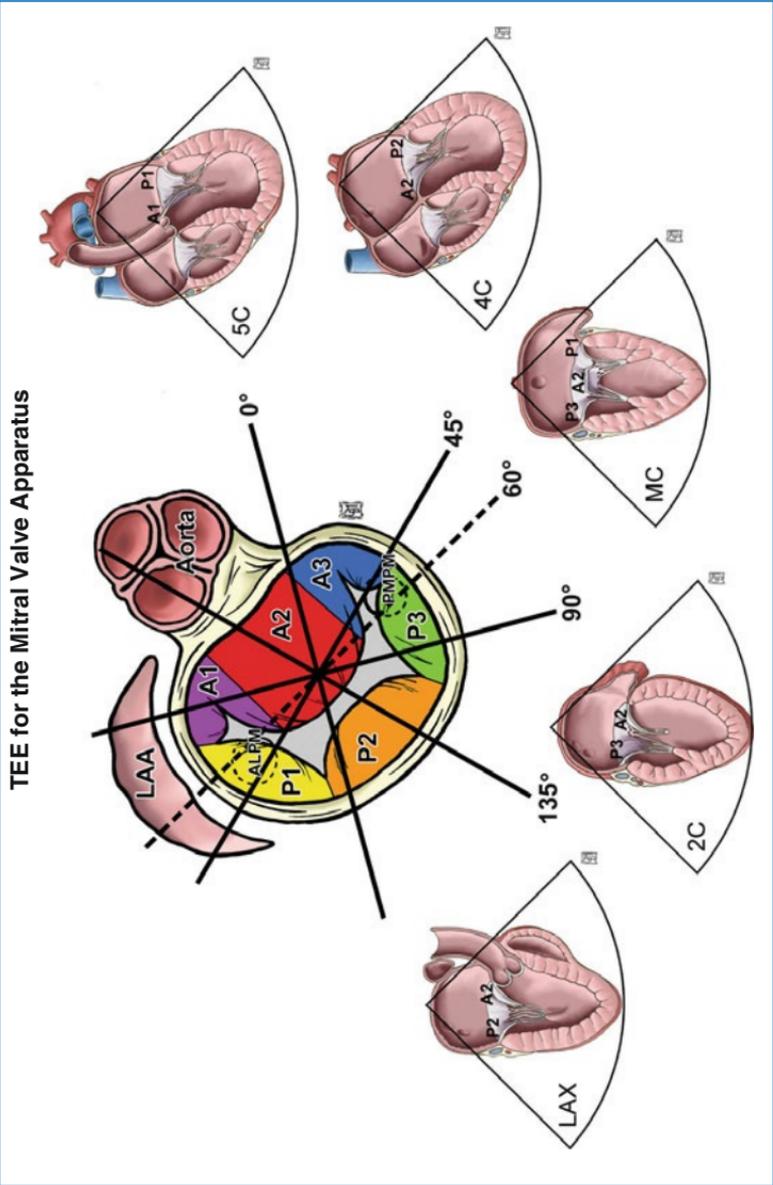
**ME 4C view (0°)**  
Specific scallops depend on:  
Anteflex (5C) view: A1/A2 + P2/P1  
Retroflex (4C) view: A3/A2 + P2/P3



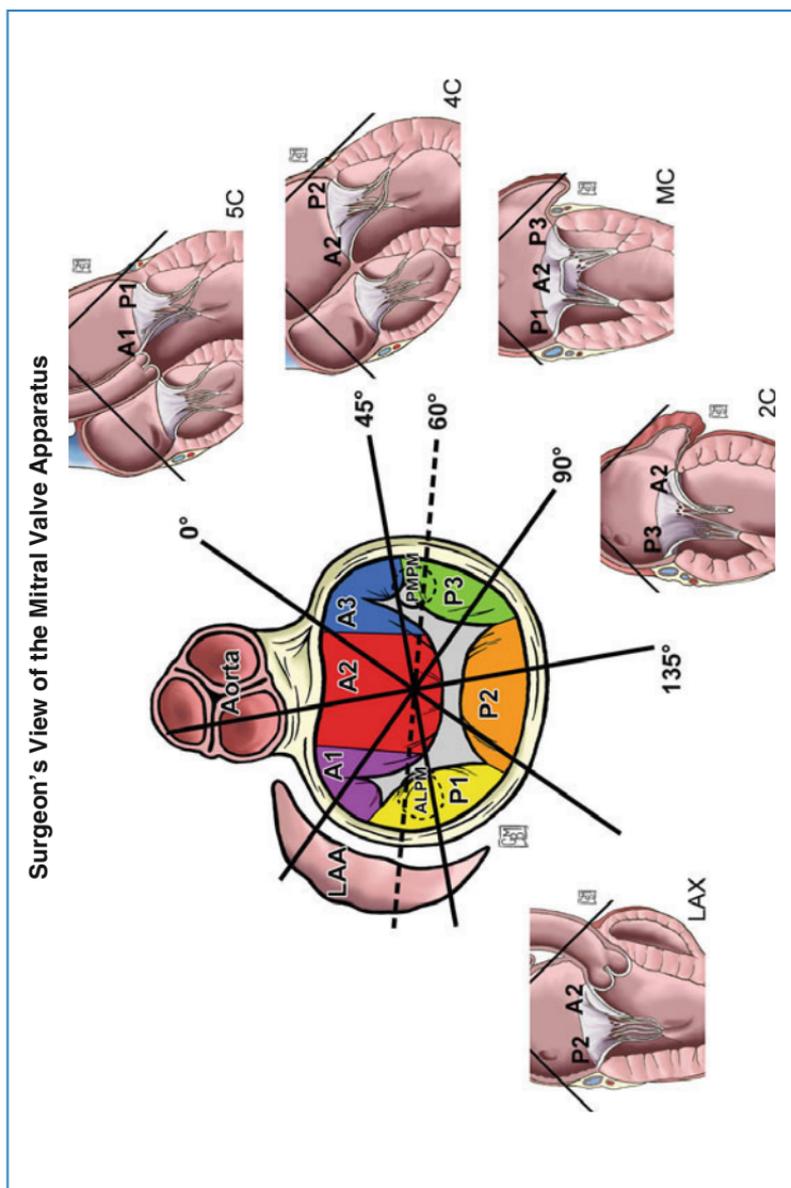
**ME 45° view**  
AMVL is on the display left  
AMVL (A3,A2) is long and coaptis  
(arrow) with short P2

Adapted from: Omran AS, et al. J Am Soc Echocardiogr 2002; 15:950-7.

TEE Views



## TEE Views



## Mitral Regurgitation

### Mitral Regurgitation

1. Etiology of regurgitation: (normal finding in 40% of patients)
  - Leaflet: prolapse, flail, myxomatous, rheumatic, endocarditis
  - Annulus: dilated (LV/LA), mitral annular calcification (MAC)
  - Chordae: rupture, elongation, shortening, tenting, SAM
  - Papillary muscle rupture, LV dysfunction
2. 2D findings:
  - Leaflets: thickened ( $> 5$  mm), calcified, malcoapt, prolapsed, flail, vegetation
  - Annulus: MAC, size (mid-diastole  $29 \pm 4$  mm)
3. Doppler findings:
  - Color: turbulent systolic flow from LV to LA, flow acceleration below MV
  - Color: jet direction: central, posterior, anterior
  - Color: area mapping (trace mosaic area), vena contracta (narrowest width), proximal flow convergence (PISA)
  - CW: systolic flow above baseline, velocity 5–6 m/s, density  $\propto$  MR, contour parabolic or early peaking, triangular shape in severe MR
  - PW: mitral inflow velocity  $> 1.5$  m/s with moderate–severe MR (no MS), A-wave predominance excludes severe MR
  - PW: pulmonary veins systolic flow reversal specific but not sensitive, absent if large LA. Eccentric jet look in contra-lateral pulmonary vein.
4. LA enlarged ( $> 55$  mm AP diameter), LA:RA ratio  $> 1$
5. LV dimensions and systolic function are important prognostic factors and indicators for surgery. Dilated due to volume overload:
  - LV size: ESD  $> 55$  mm
  - Systolic function: initially good, but worsens
6. Regurgitation severity (severe) based on the following (ASE):
  - Specific:
    - Vena Contracta:  $> 7$  mm with central jet ( $> 40\%$  LA) or any eccentric jet
    - Flow convergence (PISA):  $> 9$  mm (Nyquist 40 cm/s) central jet
    - Pulmonary vein systolic flow reversal
  - Supportive:
    - CW Doppler dense, triangular, E-wave dominant MV inflow ( $> 1.2$  m/s)
    - Enlarged LV and LA size
  - Quantitative:
    - Regurgitant volume (RegV):  $> 60$ cc
    - Regurgitant fraction (RF):  $> 50\%$
    - Effective regurgitant orifice area (EROA):  $> 0.4$  cm<sup>2</sup>

#### What to tell the surgeon

##### Pre-CPB:

- Myxomatous, calcified, prolapsed/flail segments, annular size, MAC
- Direction and severity of MR jets, pulmonary vein flow (blunted/reversed)
- LV size and function

##### Post-CPB:

- Post repair mitral leaflet morphology, Prosthetic valve function
- Residual MR, impaired mitral inflow (?stenotic)
- Complications repair: SAM, posterior wall (circumflex art), atrioventricular groove separation, AV noncoronary cusp trauma
- LV / RV function, severity TR

# Mitral Regurgitation

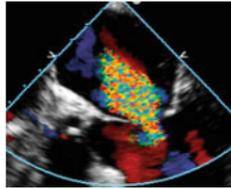
## Mitral Regurgitation Severity Assessment (AHA/ASE)

Method	Mild	Moderate	Severe
CW Doppler signal strength	Faint	Mod	Dense
Jet Area mapping (cm <sup>2</sup> ) <sup>b</sup>	< 4	4–10	> 10
Jet Area (JA) / Left Atrial (LA) area (%)	< 20	20–40	> 40
Pulmonary Venous Doppler (S wave)	Normal	Blunt	Reverse
Regurgitant Volume (cc)	< 30	30–59	≥ 60
Regurgitant Fraction (%)	< 30	30–49	≥ 50
Vena Contracta (mm) <sup>b</sup>	< 3	4–6	≥ 7
Effective Regurgitant Orifice Area (cm <sup>2</sup> )	< 0.20	0.20–0.39	≥ 0.4
PISA radius (mm) <sup>a</sup>	< 4	4–9	> 10

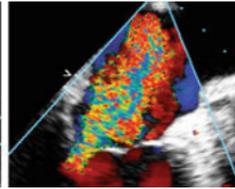
Assess regurgitation severity under physiologic conditions (SBP, afterload and LV function). Use appropriate Nyquist velocity <sup>a</sup>40 cm/s, <sup>b</sup>50–60 cm/s and color gain.  
**Adapted from: Zoghbi W, et al. J Am Soc Echocardiogr 2003;16:777-802.**

### Jet Area Mapping

- Trace mosaic jet area
- Nyquist 50–60 cm/s
- Physiology dependent
- Underestimates if eccentric jets
- Useful if multiple jets



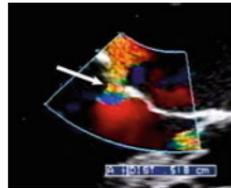
Moderate 4–10 cm<sup>2</sup>



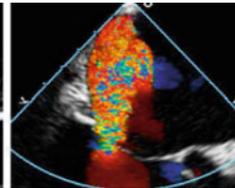
Severe > 10 cm<sup>2</sup>

### Vena Contracta

- Narrowest diameter, measure above flow acceleration region
- Nyquist 50–60 cm/s
- Useful if eccentric jets
- Not used if multiple jets
- Best measured in ME AV LAX view



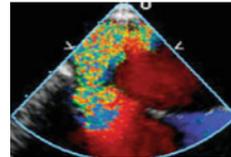
Moderate 4–6 mm



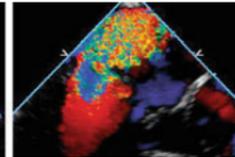
Severe > 7 mm

### PISA (EROA)

- Radius proximal flow convergence
- Nyquist 40 cm/s
- Less useful if eccentric jets
- Not used if multiple jets



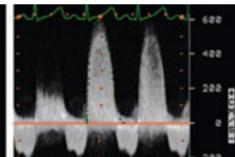
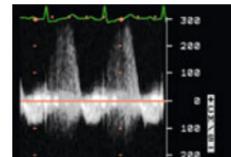
Moderate 4–10 mm



Severe > 10 mm

### CW Doppler

- Density, compare with forward flow
- Contour (complete)



# Mitral Regurgitation

## Excessive Mitral Leaflet Motion

MV annulus is saddle shaped with the highest points at 90° and 120°. Normal leaflet tip coaptation is below the annular plane in the LV. Excessive leaflet motion occurs if the MV leaflet is above the annular plane. Avoid diagnosing leaflet prolapse in only one plane and at 0° alone, examine the MV in at least two TEE planes.

### Billowing Leaflet

Part of the leaflet body is above the annulus during systole but coaptation point (arrow) is below the annulus.



### Prolapsed Leaflet

Body, leaflet tip (arrow) is above the annulus during systole without coaptation. Leaflet tips point to LV.



### Flail Leaflet

Leaflet tip is above annular plane and points towards the LA. Frequently have mobile torn chordae (arrow) attached.



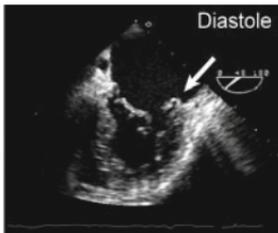
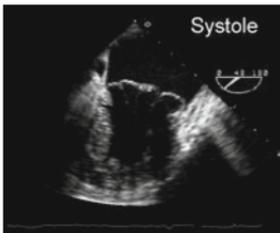
## Classic Mitral Valve Prolapse

- Defined as systolic movement of one or both mitral leaflet tips into the LA > 2 mm beyond the annular plane on TTE (parasternal LAX view), TEE is less defined.
- Posterior displacement of the coaptation point into the LA.
- May be caused by myxomatous degeneration from mucopolysaccharides deposits in the MV (see photo below).
- Associated with Marfan's, Ehlers-Danlos, SLE, WPW, and secundum ASDs.
- Concurrent other valve prolapse: tricuspid (30%), pulmonic (10%), aortic (2%).



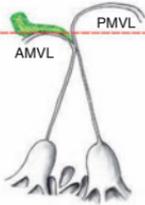
## TEE

- Thickened MV leaflets (> 4 mm)
- Systolic leaflet prolapse above annulus
- Posterior displacement of PMVL, insertion point into the LA (see arrow)
- Hinge action of the PMVL
- Mitral regurgitation results from:
  - Annular dilatation (ESD > 36 mm)
  - Chordae lengthened, redundant
  - Chordal rupture

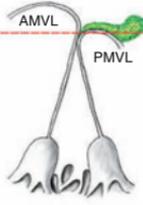


## Mitral Regurgitation

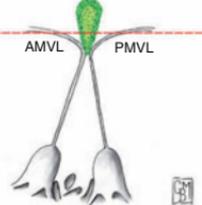
**Anterior jet (ME 4C)**



**Posterior jet (ME 4C)**



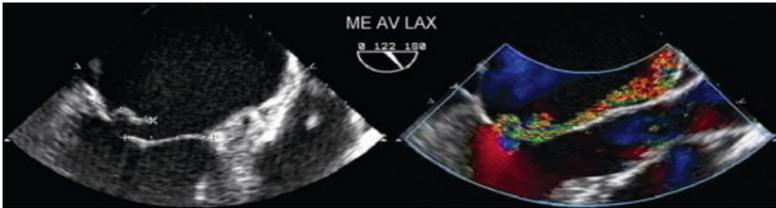
**Central jet (any ME)**



**Coanda Effect:** An eccentric MR jet may adhere to and flow around the LA wall with loss of energy. This may underestimate jet severity. Accurate assessment of MR severity depends on factors other than jet area (see pg. 91).

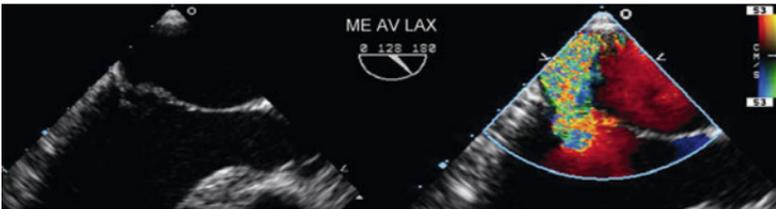
### **Anterior jet (ME AV LAX view)**

Typically prolapsed posterior leaflet, rarely restricted anterior leaflet or perforation. Jet hugs AMVL and wraps around LA towards right pulmonary veins.



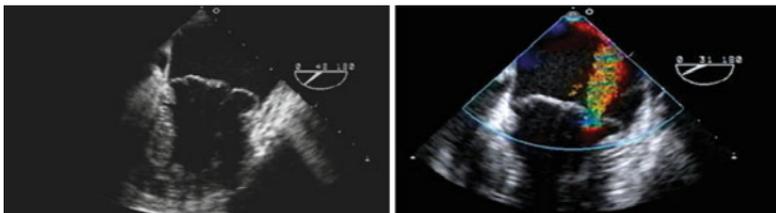
### **Posterior jet (ME AV LAX view)**

Typically prolapsed anterior leaflet, rarely restricted posterior leaflet or perforation. Jet hugs PMVL and wraps around LA towards left pulmonary veins.



### **Central jet (any ME view)**

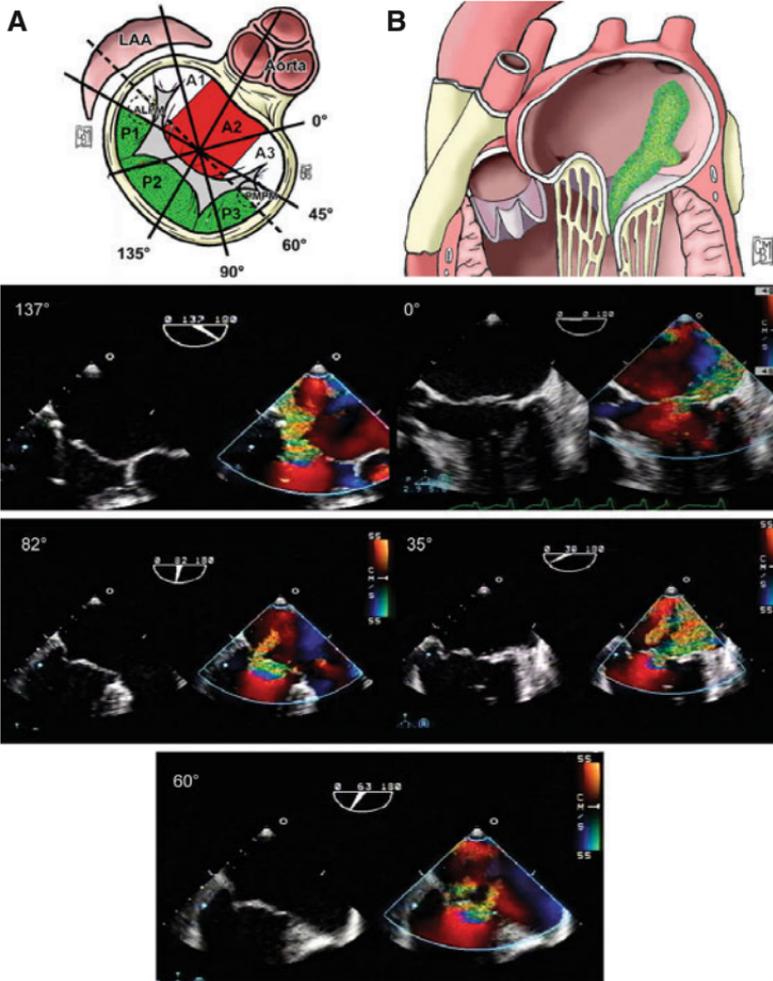
Typically bileaflet prolapse, annular dilatation, or LV dysfunction which results in bileaflet restriction. Any or all four pulmonary veins may be affected.



# Mitral Regurgitation

## Posterior Mitral Regurgitation Jet

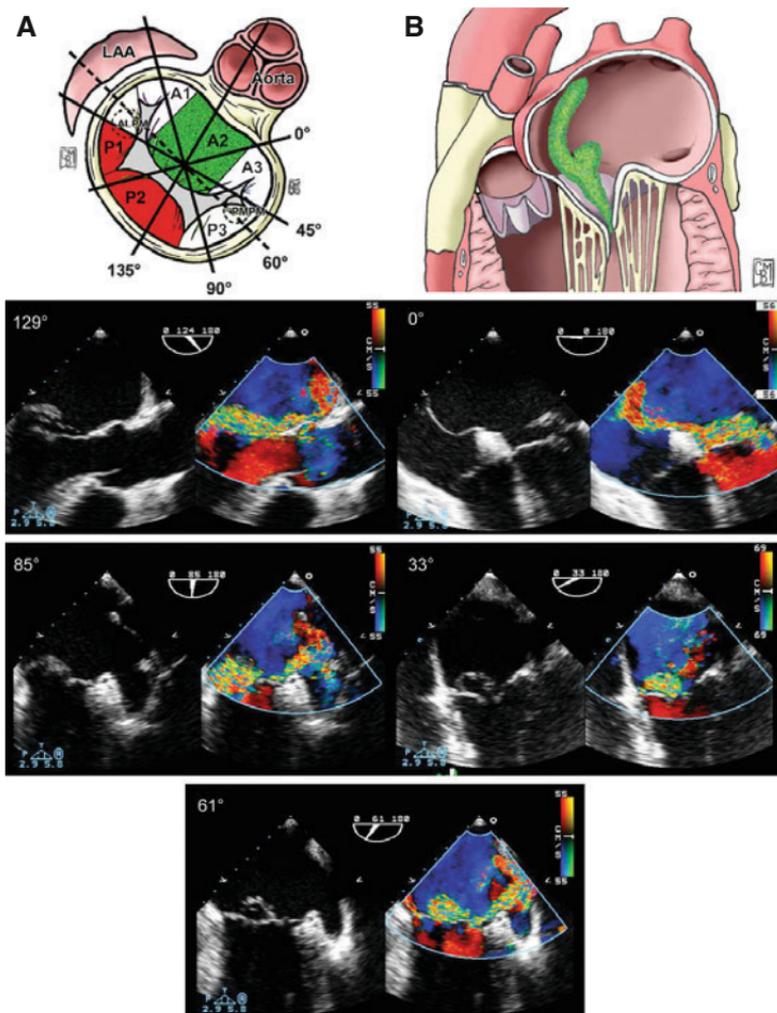
(A, B) Anterior mitral valve leaflet prolapse/flail (in red) results in a posterior directed MR jet (in green). This is shown in multiple ME views with and without color Doppler imaging (Nyquist 55 cm/s). The MR jet is posterior directed and wraps around the LA seen best at 0° (4C) and 137° (ME AV LAX). This eccentric jet is moderate to severe due to the Coanda effect. Examine and quantify the MR jet under adequate physiological conditions (BP, LV function). Abnormal left pulmonary vein flow is expected.



## Mitral Regurgitation

### Anterior Mitral Regurgitation Jet

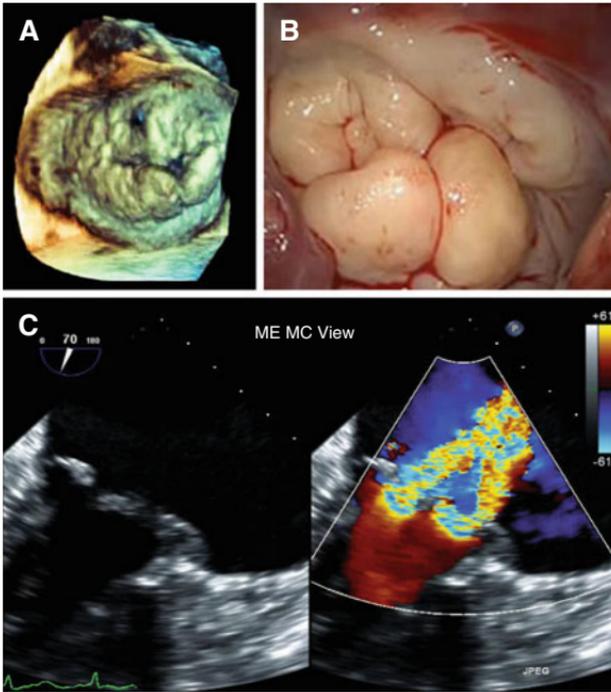
(A, B) Posterior mitral valve leaflet prolapse/flail (in red) results in an anterior directed MR jet (in green). This is shown with and without color Doppler imaging (Nyquist 69 cm/s). The MR jet is anterior directed and wraps around the LA seen best at 0° (ME 4C) and 129° (ME AV LAX). This eccentric jet is moderate to severe due to the Coanda effect. Abnormal right pulmonary vein flow is expected.



## Mitral Regurgitation

**Barlow's disease** is a degenerative disease of the MV from myxoid infiltration resulting in excessive leaflet tissue. An example is shown from the LA side (A) using 3D TEE, (B) at surgery and in the (C) 2D TEE ME Mitral Commissural view with bileaflet prolapse and severe central MR. The MV annulus is often displaced into the LA complicating the repair. (D) A 3D model reconstruction shows bileaflet prolapse.

Source: Eriksson M, et al. J Am Soc Echocardiogr 2005; 18:1014-22.



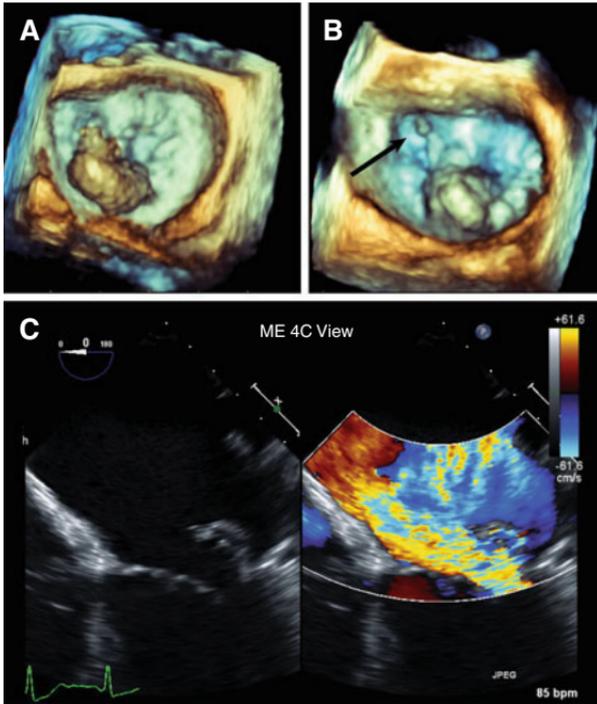
### Barlow's Mitral Valve

- Excessively thick leaflets
- Prolapse both leaflets
- Central or eccentric MR
- Annular dilatation
- Annulus displacement into LA
- Chordal elongation and thickened
- Chordal rupture uncommon
- Complex repair

## Mitral Regurgitation

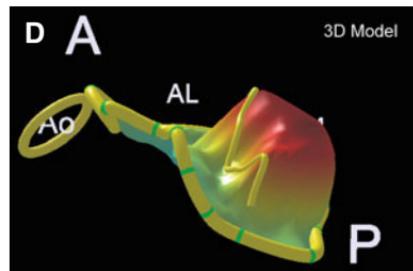
**Fibroelastic degeneration** of MV in two patients with an isolated P2 prolapse with flail tip (P2) and torn chordae (arrow). (A, B) Examples are shown from the LA side in the surgeon's orientation using real time 3D TEE. (C) Compare 2D ME 4 Chamber view of the MV with color Doppler showing severe anterior directed MR. (D) Reconstructed 3D model shows prolapsed segment.

Source: Anyanwu A and Adams D. Semin Thorac Cardiovasc Surg 2007;19: 90-96.



### Fibroelastic Disease

- Normal leaflet thickness
- Isolated segment prolapse
- Eccentric MR
- $\pm$  Annular dilatation
- No annulus displacement
- Chordal rupture
- Simple repair



## Mitral Stenosis

### Mitral Stenosis

- Etiology of stenosis
  - Valvular: rheumatic, calcific (MAC), carcinoid, SLE, congenital, drugs
  - Subvalvular: mass, myxoma
- 2D findings
  - Annulus: Ca<sup>2+</sup>, size (end diastole)
  - Leaflets: Ca<sup>2+</sup>, thickness (> 4 mm), mobility, diastolic doming "hockey stick"
  - Chordae: Ca<sup>2+</sup>, thickened, extent of subvalvular involvement
  - Planimeter MVA in TG basal SAX view (underestimate MVA)
- Doppler findings
  - Color: turbulent diastolic flow, proximal flow acceleration
  - PW/CW: peak velocity > 3 m/s, peak/mean P gradient
    - Note elevated transmitral inflow also occurs with high cardiac output, MR and restrictive diastolic filling
  - Pressure half-time (PHT) for native MVA
- Stenosis severity (severe)
  - Peak velocity > 3m/s
  - Mean pressure gradient > 10 mmHg
  - Mitral valve area < 1.0 cm<sup>2</sup> (2D planimetry, PHT)
- Coexisting MR (overestimates measured pressure gradients)
- LA enlargement (LAX view: A-P diameter > 45 mm), smoke, thrombus in LAA
- PASP (estimate from TR jet)
- Coexisting TR severity
- RV function: dilated, hypertrophy, IVS paradoxical motion
- LV function: small underfilled, SWMA (postero-basal segment)

### Severity Assessment (EAE/ASE Guidelines)

	Valve area (cm <sup>2</sup> )	Mean gradient (mmHg)	PHT (msec)	Peak pulmonary artery P (mmHg)
Normal	4–6		40–70	20–30
Mild	> 1.5	< 5	70–150	< 30
Moderate	1.0–1.5	510	150–200	30–50
Severe	< 1.0	> 10	> 220	> 50

If have associated moderate–severe MR, the peak velocity and transmitral pressure gradients are overestimated so need to calculate valve area. In NSR 60–80 bpm  
**Adapted from: Baumgartner H, et al. J Am Soc Echocardiogr 2009;22:1-23.**

#### What to tell the surgeon

##### Pre-CPB:

- Calcific vs rheumatic valve
- Chordal involvement
- Annulus size (29 ± 4 mm)
- Mitral annular calcification (MAC)
- LA size (severe > 50 mm), LAA thrombus
- RV function, TR severity

##### Post-CPB:

- Peak/mean Pressure gradients
- Residual MR
- Prosthetic function

## Mitral Stenosis

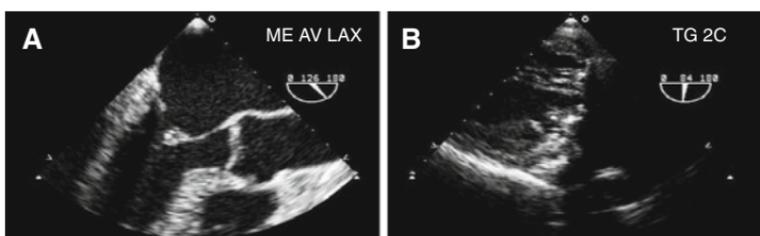
Mitral Stenosis	2D echo	Color/spectral Doppler
ME 4C (0°) ME commissural (60°) ME 2C (90°) ME AV LAX (120°)	Annulus: Ca <sup>2+</sup> , size Leaflets: Ca <sup>2+</sup> , thick, mobility Chordae: Ca <sup>2+</sup> , thick	Turbulent diastolic flow PISA MV inflow pattern: peak/mean Pressure half time (PHT)
TG SAX (0°)	Ca <sup>2+</sup> , planimetry	Commissural origin color
TG LAX (90°)	Subvalvular apparatus	

### Grading of MV Characteristics in Mitral Stenosis

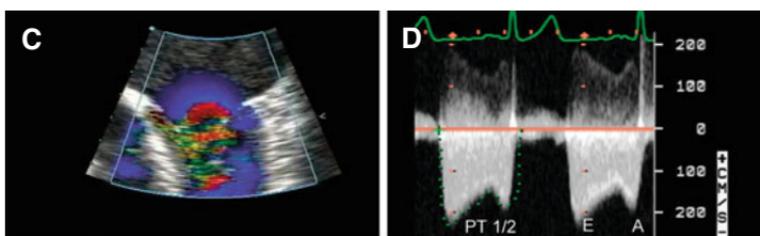
Grade	Mobility	Leaflet thickened	Subvalvular	Calcification
1	Tips restricted	4–5 mm	Minimal	Minimal
2	Base-mid normal	5–8 mm	1/3 chordae	Leaflet margins
3	Base normal	5–8 mm	2/3 chordae	Mid leaflet
4	No movement	> 8–10 mm	Total	Majority leaflet

The echo score quantifies the severity of the rheumatic MV morphologic derangement to establish a predictor of outcome after percutaneous balloon valvuloplasty. Valve score < 8 has a good outcome. Increased score associated with suboptimal outcome, ↑ mortality, restenosis, heart failure, ↑ need for cardiac surgery.

Source: Wilkins G. Br Heart J 1988; 60:300.



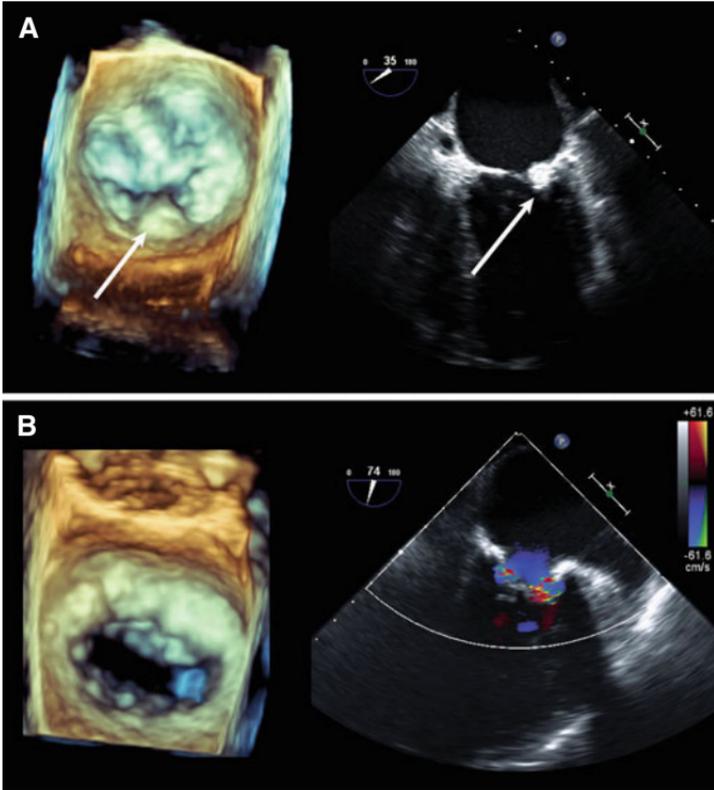
(A) Fusion of the rheumatic leaflet edges with elevated LAP pushes the more mobile body of the AMVL toward the LV producing diastolic doming of the AMVL giving it a *hockey stick* appearance. (B) The subvalvular chordae, best seen in the TG 2C view, are short and thickened resulting in restricted leaflet motion.



(C) Color Doppler shows proximal flow acceleration and turbulent antegrade flow through the stenotic MV. (D) Spectral Doppler (PW/CW) can be traced to measure the peak and mean pressure gradients and analyzed for the pressure half-time (PT1/2) to estimate the MV area (see pg. 46).

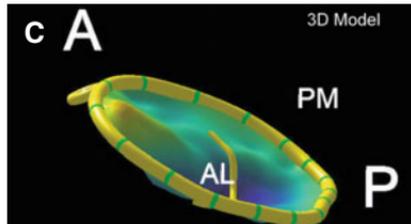
## Mitral Stenosis

Patients with restricted MV leaflet motion from annular calcification. (A) This patient has an isolated block of calcium (arrow) extending from the annulus into the P2 segment of the MV. This is shown in 3D TEE from the LA side in the surgeon's orientation compared with a 2D ME view. (B) This patient has severe annular calcification, with shadowing in 2D, in the setting of aortic stenosis. (C) Reconstructed 3D model shows restriction below the annular plane of the posterior MV leaflet.



### Restricted Mitral Valve

- Thick leaflets, calcium
- No prolapse
- Restricted mobility (open, close)
- Annular calcium
- Chordal restriction
- Difficult repair
- MV replacement



## Normal Values

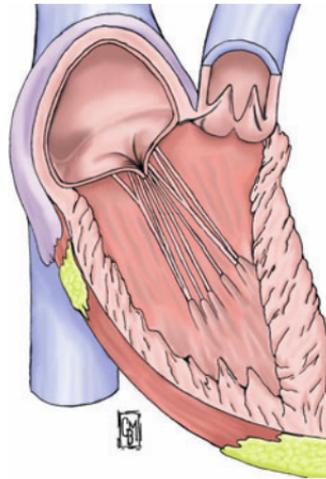
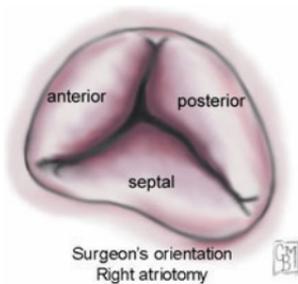
2D Exam Normal Values [Mean ± SD]	
<b>Left Ventricle, diameters</b>	
Antero-posterior, diastole	43 ± 7 mm
Antero-posterior, systole	28 ± 6 mm
Medio-lateral, diastole	42 ± 7 mm
Medio-lateral, systole	27 ± 6 mm
<b>Left Atrium (end systole)</b>	
Antero-posterior diameter	38 ± 6 mm
Medio-lateral diameter	39 ± 7 mm
Appendage, length	28 ± 5 mm
Appendage, diameter	16 ± 5 mm
Pulmonary vein	11 ± 2 mm
<b>Right Atrium (end systole)</b>	
Antero-posterior diameter	38 ± 5 mm
Medio-lateral diameter	38 ± 6 mm
<b>Right Heart Structures</b>	
Superior vena cava	15 ± 3 mm
Coronary sinus	6.6 ± 1.5 mm
Right ventricular outflow tract	27 ± 4 mm
Mean PA	20 ± 5 mm
Right pulmonary artery	17 ± 3 mm
<b>Aorta, Thoracic</b>	
Root	28 ± 3 mm
Proximal descending	21 ± 4 mm
Distal descending	20 ± 4 mm
Reference values for normal adult transesophageal echocardiographic measurements in 60 normal patients Source: Cohen GI, et al. J Am Soc Echocardiogr 1995;8:221-30.	

Normal Valve Values				
	Annulus <sup>a</sup> (mm)	Valve area (cm <sup>2</sup> )	Velocity (m/s)	Peak gradient (mmHg)
Aortic	21 ± 3	3.0–4.0	1.4–2.2	8–20
Mitral <sup>b</sup>	27 ± 4	4–6	< 0.9	< 4
Pulmonic	21 ± 3	2.5–3.5	< 1.0	< 4
Tricuspid	28 ± 5	7–9	< 0.7	< 2
<sup>a</sup> Annulus diameter changes size during the cardiac cycle and is largest when the valve is open. The measurements shown here are in systole <sup>b</sup> The mitral valve annulus measured at end diastole (open) is larger 29 ± 3, then during systole (closed) 27 ± 4. The mitral annulus is ellipsoid in shape and should be measured in two views, 0° (smaller) and 90° (larger)				

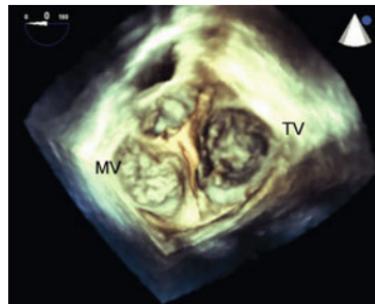
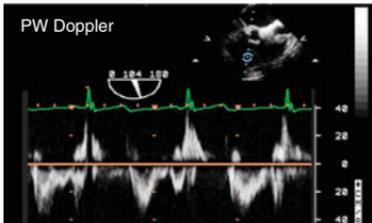
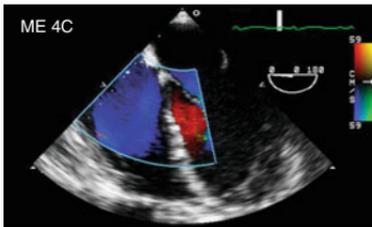
# Tricuspid Valve Anatomy

## Tricuspid Valve Anatomy

- Annulus: fibrous ring to which leaflets attach  
 hinge point is apically displaced below MV annulus (ME 4C view)  
 distensible size (diameter end systole  $28\text{ mm} \pm 5$ )  
 TV area  $7\text{--}9\text{ cm}^2$
- Three valve leaflets: (size varies)  
 septal > anterior > posterior
- Three commissures: anteroseptal, anteroposterior, posteroseptal
- Chordae: support leaflets during systole, attach to papillary muscles and directly to septal wall (unlike MV)
- Three papillary muscles:  
 anterior, posterior,  $\pm$  septal



**TV normal color and spectral Doppler**  
 TV has diastolic laminar flow (blue) with the lowest velocity ( $< 70\text{ cm/s}$ ).



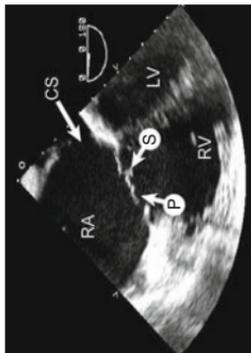
## Base of the Heart

A 3D full volume image of the base of the heart shows the relationship of all four cardiac valves. The three leaflets of the normal tricuspid valve are shown closed during systole and are comparable to the diagram on the next page. The TV has the largest valve orifice area.

## TEE Views

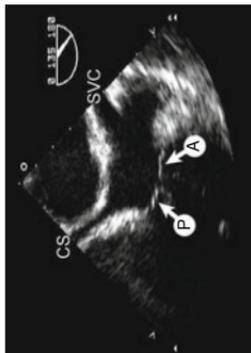
### Coronary Sinus (CS) View (0°)

Advance probe from 4C view to GE junction, see TV and inflow from CS.



### Modified Bicaaval View (110°–140°)

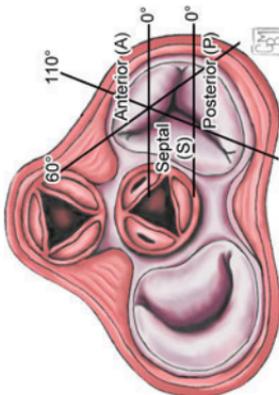
Increase angle from 90° bicaaval view, TV centered, good Doppler alignment.



### TG SAX View (0°–40°)

All three leaflets imaged simultaneously  
Poor Doppler alignment

### Tricuspid Valve Systole



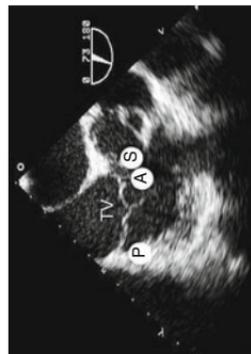
### ME 4C View (0°)

Annular dimensions (28 ± 5 mm)  
TR direction + trace extent into RA



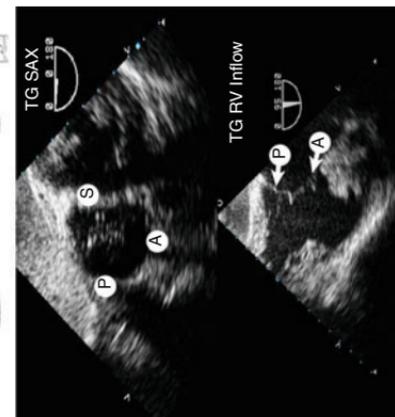
### ME RV inflow-outflow View (60°–75°)

(posterior/left, anterior or septal/right)  
Better Doppler alignment



### TG RV Inflow View (90°–120°)

Subvalvular apparatus, chordae  
Poor Doppler alignment



## Tricuspid Regurgitation

### Tricuspid Regurgitation

#### 1. Etiology of regurgitation:

- Physiological TR occurs in > 90% of patients
- Annulus: dilated from high PAP (MS, MR, Eisenmenger's, cor pulmonale)
- Valvular: prolapse, rheumatic, carcinoid, myxomatous, endocarditis
  - carcinoid: thickened, shortened immobile leaflets
  - rheumatic: thickened leaflets, TR > TS
- Ebstein's anomaly: TV leaflets (septal) apically displaced (see p. 172)
- Catheter, pacer

#### 2. 2D findings:

- Leaflets: thickened, calcified, prolapse, malcoaptation, flail
- Annulus: dilated > 34 mm end-systole (normal < 28 mm)

#### 3. Doppler findings:

- Color: turbulent (mosaic) retrograde flow, jet direction is usually toward IAS, laminar (red) retrograde flow if severe RV failure
- Color: area, vena contracta (proximal jet width), PISA radius
- CW: systolic flow towards transducer, peak velocity unrelated to TR severity
- PW: hepatic vein flow systolic reversal is 80% sensitive
- PW: TV inflow ↑ E wave velocity > 1 m/s

#### 4. Associated findings:

- RA, RV dilated
- Paradoxical IVS motion (volume overload), IAS bulges to left "D" shape
- Dilated IVC (> 2 cm) and hepatic vein (> 1 cm)

#### 5. Regurgitation severity:

- Color map area: for central jet, invalid with eccentric jets, not sole parameter
- Hepatic vein systolic flow reversal: may be absent in chronic TR if RA dilated
- IVC > 2cm, no respiratory variation, normal IVC if acute TR
- CW density and contour: dense triangular with early peaking is severe

### Tricuspid Regurgitation Severity (ASE/ACC/AHA)

	Mild	Moderate	Severe
RV/RA/IVC size	Normal	Normal or dilated	Dilated
Jet area (cm <sup>2</sup> ) <sup>a,c</sup>	< 5	5–10	> 10
VC width (cm) <sup>a</sup>	Not defined	Not defined, but < 0.7	> 0.7
PISA (cm) <sup>b</sup>	≤ 0.5	0.6-0.9	> 0.9
CW jet density	Soft, parabolic	Dense, variable shape	Dense, triangular
Hepatic vein flow	S dominance	S blunting	S reversal
Nyquist limit: <sup>a</sup> (50–60 cm/s), <sup>b</sup> (28cm/s); <sup>c</sup> not valid with eccentric jets; S = systolic			
Adapted from Zoghbi W, et al. J Am Soc Echocardiogr 2003;16:777-802.			

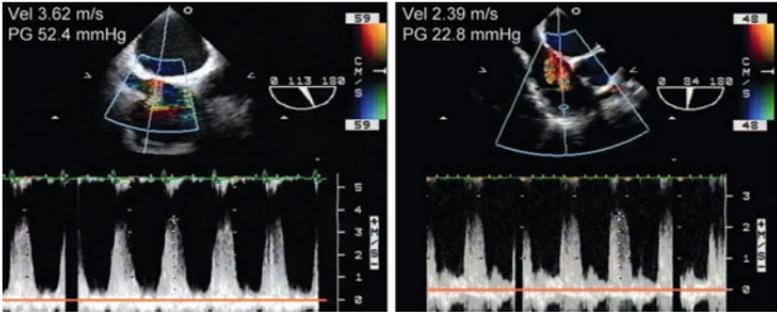
#### What to tell the surgeon

- Leaflet morphology: myxomatous, prolapse, endocarditis
  - Annulus size in systole (28 ± 5 mm)
  - TR jets number and direction, severity (color map area/ RA area)
- Post-CPB:
- Annulus size
  - TR severity
  - TV inflow (? stenosis)

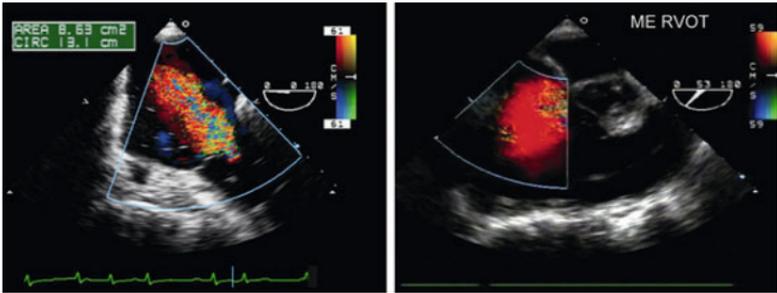
## Tricuspid Regurgitation

TR CW Doppler trace indicates flow between the RA and RV across a closed TV. Adding RAP to the measured peak TR pressure estimates the RVSP (or PA systolic pressure, see pg.42). Note the TR peak pressure gradient is not a measure of TR severity, but estimates pulmonary artery pressure. A laminar TR jet may underestimate RVSP as the RA and RV act as a single chamber.

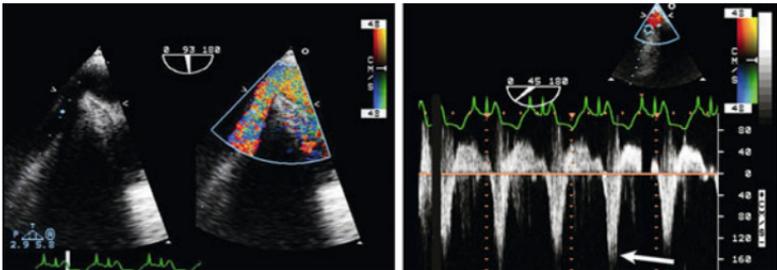
Similar dense TR spectral Doppler traces have different peak velocities. The higher velocity indicates pulmonary hypertension (52 mmHg) compared with normal PASP.



Color flow mapping of moderate–severe TR jet appears mosaic in the presence of adequate RV function and laminar with severe RV dysfunction.



Systolic reversal of hepatic vein flow in severe TR is seen with color Doppler (mosaic color) and PW Doppler S-wave (arrow) in this hepatic vein view.



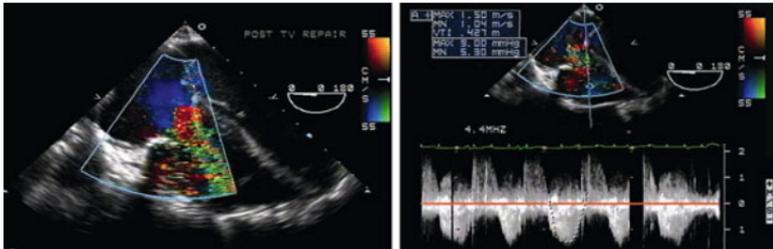
# Tricuspid Stenosis

## Tricuspid Stenosis

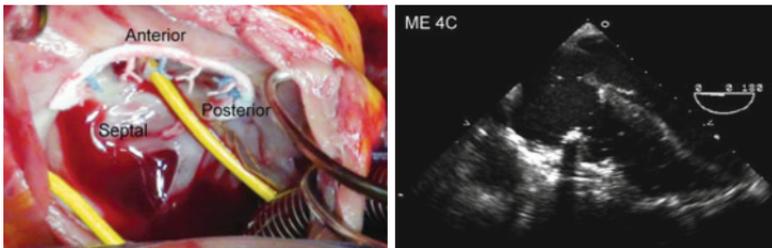
1. Etiology of stenosis:
  - Valvular: rheumatic (+mitral), carcinoid (+pulmonic)
  - Obstruction: tumor, vegetation, thrombus, extra-cardiac compression
2. 2D findings:
  - Leaflets: thickened
  - Decreased leaflet mobility, tethered leaflet tips (diastolic doming)
3. Doppler findings:
  - Color: turbulent diastolic flow, may also have TR (systolic flow)
  - CW: HR between 70 and 80, TV inflow peak E velocity > 1.0 m/s  
mean Pressure gradient
    - Mild < 2 mmHg
    - Moderate 2–5 mmHg
    - Severe > 5 mmHgCW for PHT of TV area (TVA)
4. Associated findings: RA enlarged, IVC dilated (> 2.3 cm)
5. Stenosis severity (severe), ASE guidelines<sup>a</sup>
  - TV area < 1.0 cm<sup>2</sup>
  - peak velocity > 1.5 m/s, mean pressure > 5 mmHg, VTI > 60 cm
  - PHT valve area is not validated (use TVA = 190/PHT), continuity, PISA

<sup>a</sup>Adapted from Baumgartner H, et al. *J Am Soc Echocardiogr* 2009;22:1-23.

Turbulent diastolic color flow through the TV with proximal flow acceleration and a CW Doppler mean gradient > 5 mmHg suggests severe tricuspid stenosis.



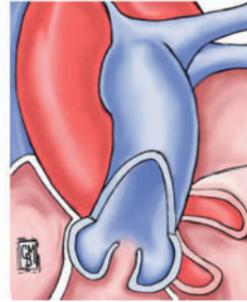
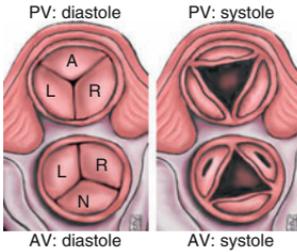
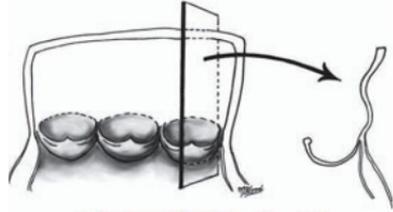
Tricuspid valve with an annuloplasty ring exposed through a right atriotomy at the time of implantation. Tricuspid annuloplasty ring is seen as bright echo with shadowing in a rotated ME 4C view at 0°.



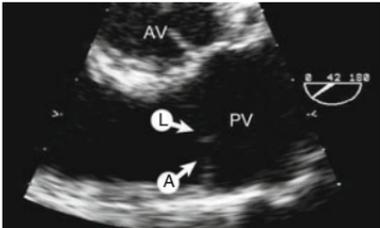
# Pulmonic Valve

## Pulmonic Valve Anatomy

- Anterior cardiac structure
- Difficult to image with TEE
- Valve: Three semi-lunar cusps:
  - Right (R), Left (L), Anterior (A)
- PA: slightly dilated forming sinus
- AV and PV normally lie at 90° planes to each other. ME RVOT view images AV SAX (PV LAX) and in ME AV LAX view the PV is in SAX though difficult to see as it is anterior.



## Pulmonic Valve TEE Views



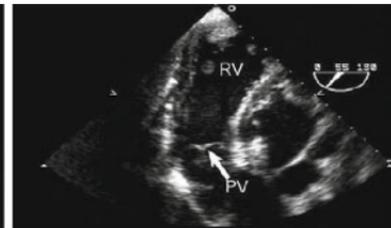
**ME RV Inflow-Outflow View (45–60°)**  
 Difficult to see cusps, try zoomed view  
 Measure PV annulus ( $21 \pm 3$  mm)



**ME Ascending Aortic SAX View (0°)**  
 Useful for Doppler alignment of PA flow  
 Diameter main PA:  $20 \pm 5$  mm



**UE Aortic Arch SAX View (60–90°)**  
 Cusp morphology, measure annulus  
 Doppler alignment for PV or PA flow



**TG RV Modified View (30–60°)**  
 Useful Doppler alignment  
 Normal PV peak velocity 0.5–1.0 m/s

# Pulmonic Insufficiency

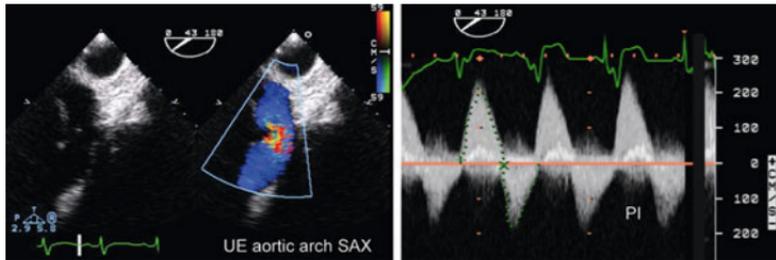
## Pulmonic Insufficiency

- Etiology of insufficiency:
  - Physiologic PI in 80% patients
  - Valvular: myxomatous, Marfan's, congenital, endocarditis, prosthetic
  - Dilated PA, RVOT, ↑ PA pressures
  - Carcinoid
- 2D findings:
  - Difficult to image cusps well as PV is an anterior structure
  - PV annulus or PA dilated
- Doppler findings:
  - Color: blue or turbulent diastolic flow in RVOT, may be brief in duration
  - PW/CW: diastolic flow away from baseline, density, and deceleration slope
  - PW PV flow: ↑ peak systolic velocity, compare with systemic (AV) flow
- Associated findings: RV dilated, posterior displacement of LV septum
- Severity of insufficiency is difficult to quantify
  - Mild PI is common, Swan-Ganz only causes mild PI
  - Color/ Spectral Doppler flow reversal in main PA (see below)

PI Severity (ASE <sup>a</sup> )	Mild	Moderate	Severe
Morphology	Normal	Normal, abnormal	Abnormal
RV size	Normal	Normal or dilated	Dilated
Jet size <sup>b</sup>	Thin, < 10 mm length	Intermediate	Large, wide origin
CW density	Soft	Dense	Dense
Deceleration slope	Slow	Variable	Steep <sup>c</sup>
PA:systemic flow	Slight increase	Intermediate	Greatly increased

<sup>a</sup>Adapted from: Zoghbi W et al. J Am Soc Echocardiogr 2003;16:777-802.  
<sup>b</sup>Nyquist limit (50–60 cm/s), <sup>c</sup>Steep deceleration not specific for severe PI

Severe PI has holodiastolic flow reversal in main PA by color Doppler (blue) with equally dense forward and reverse CW Doppler flow in UE Aortic Arch SAX view.



What to tell the surgeon

Pre-CPB:

- Valve morphology: calcified, prolapse, endocarditis, prosthetic failure
- PA dilated: > 20 mm
- Difficulty to quantify severity, color, and spectral Doppler

Post-CPB:

- Prosthetic valve function: peak, mean gradients, paravalvular leaks

# Pulmonic Stenosis

## Pulmonic Stenosis

### 1. Etiology of stenosis:

- Normal pulmonic valve area  $2 \text{ cm}^2/\text{m}^2$
- Valvular: rheumatic, carcinoid, prosthetic
- Congenital
- Infundibular (RV hypertrophy)

### 2. 2D findings:

- Valve: thickened, calcified, immobile, systolic doming
- RVOT narrowed in infundibular PS
- RVH  $> 5 \text{ mm}$  thick (pressure overload), RV dilated
- Post-stenotic PA dilatation ( $> 20 \text{ mm}$ )

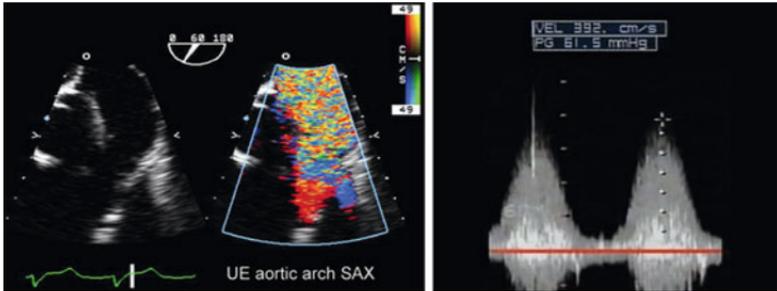
### 3. Doppler findings:

- Color: turbulent systolic flow at level of obstruction, also may have PI
- PW to locate level of obstruction (valvular, subvalvular)
- CW velocity and peak Pressure gradients (ASE<sup>a</sup>)
  - Overestimate gradients if PI
  - Mild:  $< 3 \text{ m/s}$ ,  $< 36 \text{ mmHg}$
  - Moderate:  $3\text{--}4 \text{ m/s}$ ,  $36\text{--}64 \text{ mmHg}$
  - Severe:  $> 4 \text{ m/s}$ ,  $> 64 \text{ mmHg}$
- PASP does not equal RVSP in the presence of PS
  - PASP = RVSP (from TR + RAP) – PV pressure gradient

### 4. Stenosis severity (severe by ASE guidelines<sup>a</sup>)

- Peak velocity  $> 4 \text{ m/s}$
- Peak gradient  $> 64 \text{ mmHg}$
- Continuity equation for valve area ( $< 0.5 \text{ cm}^2$ )

<sup>a</sup>Adapted from: Baumgartner H, et al. J Am Soc Echocardiogr 2009;22:1-23.



### What to tell the surgeon

#### Pre-CPB:

- Valve morphology: prosthetic failure, calcified
- Poststenotic PA dilatation  $> 20 \text{ mm}$
- Annulus size  $21 \pm 3 \text{ mm}$
- Stenosis severity: peak, mean pressure gradients

#### Post-CPB:

- Prosthetic valve function: peak, mean gradients, paravalvular leaks

# Prosthetic Valves Transcatheter Valves and Valve Repairs

Overview .....	118
Mechanical Valves .....	119–121
Bioprosthetic Valves .....	122
Patient Prosthetic Mismatch .....	123
Prosthetic Valve Function .....	124–127
Transcatheter Valves .....	128–131
Mitral Valve Repairs .....	132–133
Aortic Valve Repairs .....	134–135

## Overview

Types of Prosthetic Valves	
<b>Tissue (Bioprosthetic)</b> Stented porcine: Hancock, CE, Mosaic Stented bovine: Ionescu-Shiley, CE Stentless porcine: SPV, Freestyle Homograft: aortic, mitral	<b>Mechanical</b> Caged Ball: Starr-Edwards Tilting disc: Bjork-Shiley, Medtronic-Hall Bileaflet: St. Jude, CarboMedics Valved conduit: St.Jude, Medtronic-Hall

### Normal Prosthetic Valve Findings

Aortic homograft	Antegrade flow similar to native valve Thickened aortic annulus/root, no acoustic shadow None to trivial valve regurgitation
Tissue valve	Antegrade flow similar to native valve Three struts, acoustic shadow Mild valvular regurgitation
Caged ball	Antegrade flow through valve periphery To avoid cage acoustic shadowing image in LAX None to trivial valve regurgitation around "ball"
Tilting disc	Antegrade flow through two orifices (major and minor) Single disc, acoustic shadowing Two to three regurgitant washing jets: large central + smaller peripheral
Bileaflet	Antegrade flow through three orifices Bileaflet motion, acoustic shadowing Three regurgitant washing jets: one central and two peripheral

### Prosthetic Valve Pressure Gradients (PG)

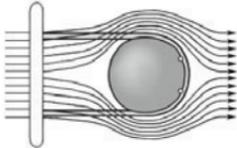
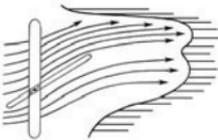
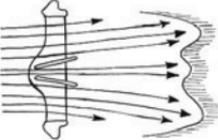
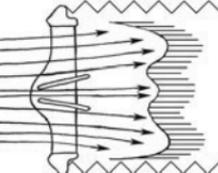
Type	Mitral			Aortic <sup>a</sup>		
	Vmax (m/s)	Pmax (mmHg)	Pmean (mmHg)	Vmax (m/s)	Pmax (mmHg)	Pmean (mmHg)
Starr-Edwards	1.9±.4	14±5	5±2	3.2±.6	38±11	23±8
St. Jude <sup>a</sup>	1.6±.3	10±3	4±1	2.4±.3	25±5	12±6
Bjork-Shiley	1.6±.3	10±2	3±2	2.5±.6	23±8	14±5
CE	1.8±.2	12±3	6±2	2.5±.5	23±8	14±5
Hancock	1.5±.3	9±3	4±2	2.4±.4	23±7	11±2
Stentless	None	None	None	2.2	19	3±1

- <sup>a</sup> PG varies with valve size (aortic position): 19 mm (20 mmHg), 23 mm (12 mmHg)
- Pressure recovery overestimates St Jude AVR gradient
- Valve sizes describe the outer valve diameter, not the internal orifice diameter
- Prosthesis patient mismatch: normal prosthesis function with high transvalvular gradient (see pg. 123)

#### What to tell the surgeon Post-CPB:

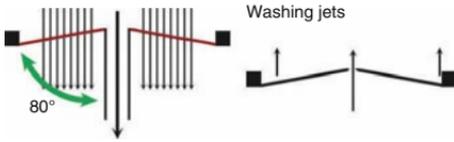
- Valve well seated
- Leaflets mobile (2D and color Doppler)
- Valvular functional leaks (washing jets, physiologic)
- Paravalvular leaks (color Doppler)
- Peak and mean valve pressure gradients
- Effective orifice area (aortic valve)
- Obstruction LVOT (MV strut), SAM of AMVL (if AV prosthesis too small)

## Mechanical Valves

Mechanical Valves		
Valve type	Flow through valve	Echocardiographic findings
<p>Starr-Edwards (discontinued 2007)</p> 	 <p>Photo courtesy of Edwards Lifesciences Irvine, California</p>	<p><b>Ball Cage</b></p> <ul style="list-style-type: none"> <li>• Ball larger than orifice</li> <li>• Turbulent antegrade flow through valve periphery</li> <li>• High profile</li> <li>• Small orifice, high pressure</li> <li>• ↑ thromboembolism risk</li> <li>• Trivial valve regurgitation</li> <li>• No washing jets</li> </ul>
<p>Medtronic-Hall (below), Bjork-Shiley (discontinued)</p> 	 <p>Photo courtesy of Medtronic</p>	<p><b>Tilting Disc</b></p> <ul style="list-style-type: none"> <li>• Single disc + eccentric strut/hinge</li> <li>• Opening angle 60°–70°</li> <li>• Back pressure on larger disc portion closes disc</li> <li>• Two antegrade flow orifices across valve (major, minor)</li> <li>• Three washing jets large central jet and smaller jets around occluding disc and sewing ring</li> </ul>
<p>St. Jude (below), Carbomedics</p> 	 <p>Photo courtesy of St Jude Medical</p>	<p><b>Bileaflet</b></p> <ul style="list-style-type: none"> <li>• Two symmetrical leaflets + two hinges, low profile</li> <li>• Bileaflet pivot motion, opens to 80°</li> <li>• Antegrade flow through three orifices</li> <li>• Less obstructive, lowest P</li> <li>• Three regurgitant washing jets: one central + two peripheral</li> <li>• Most regurgitant fraction (10%)</li> </ul>
<p>St. Jude (below), Medtronic-Hall</p> 	 <p>Photo courtesy of St Jude Medical</p>	<p><b>Valved Conduit</b></p> <ul style="list-style-type: none"> <li>• Typically mechanical valve attached to Dacron conduit</li> <li>• Sewn in as single unit</li> <li>• Cannot have paravalvular leak, as all leaks would appear outside the heart</li> <li>• Washing jets depend on valve type</li> </ul>

# Mechanical Valves

## Mechanical Bileaflet

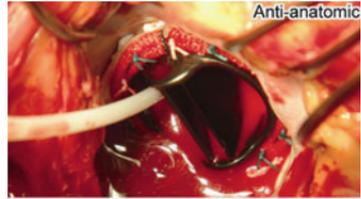


- Pressure opens leaflets (80° arc), backpressure closes leaflets.
- Outer two large orifices.
- Washing (regurgitant) jets prevent blood stasis.

## Mitral Position

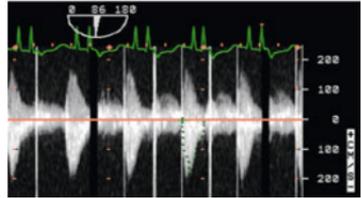
Mechanical valve is orientated to minimize disc entrapment by submitral chordae:

- Anti-anatomic: major orifice towards IVS, common for single disc valves, bileaflet valves, best seen in ME 120° view
- Anatomic: pivot points normally where commissures are, seen best in 0° view

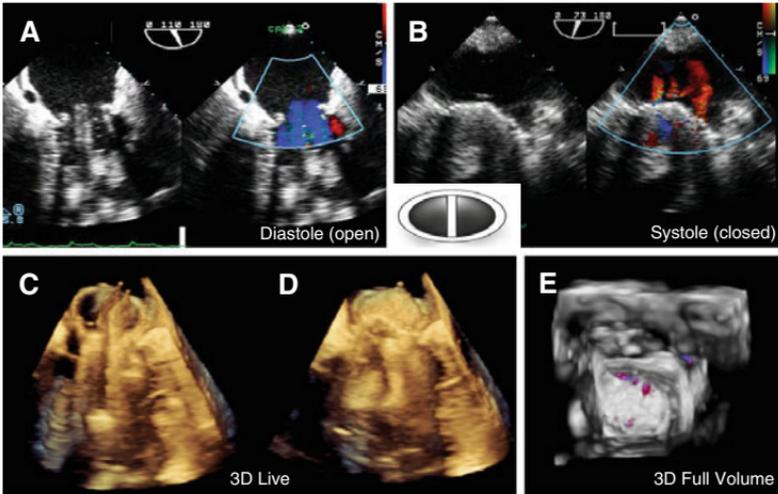


## TEE Assessment

- Easily imaged in all ME mitral views
- Symmetric leaflet mobility (open/close)
- Two to three washing jets
- Peak/mean Pressure gradients
- Paravalvular leak outside sewing ring



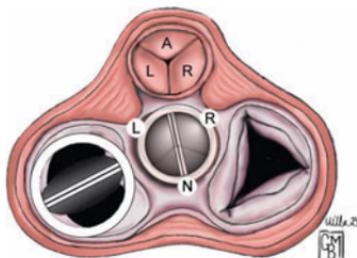
Mechanical bileaflet valve shown (A) open during diastole with laminar flow and (B) closed (valve inset) during systole with washing jets. The valve is imaged with 3D Live mode (C) open, (D) closed, and with (E) 3D full volume color Doppler showing washing jets at the hinge points.



## Mechanical Valves

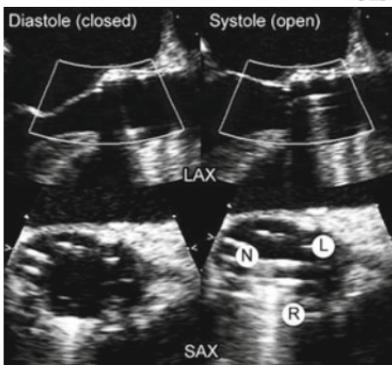
### Aortic Position

In the aortic position, the valve is orientated with one pivot between the LCC and RCC. This allows for smooth opening of the valve discs without obstructing the coronary arteries. Mechanical valves are seldom used in the tricuspid or pulmonic position, as insufficient pressure exists to open and close the valve.



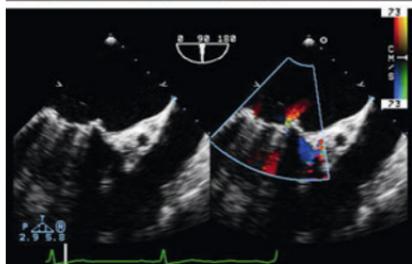
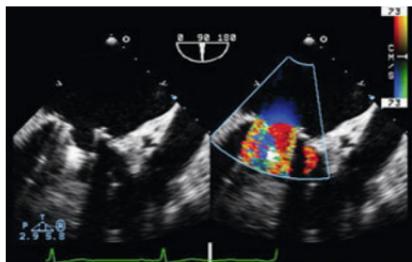
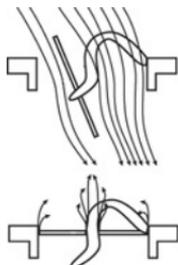
### TEE Assessment

- ME AV LAX shadowing, use TG views
- Leaflet mobility (TG, ME AV SAX)
- Washing jets (ME AV LAX/SAX, TG)
- Peak/mean pressure gradients (TG)
- Paravalvular leaks outside sewing ring (TG, ME AV LAX/SAX)



### Mechanical Tilting Disc

- Single disc + eccentric strut/hinge
- Opening angle  $60^{\circ}$ – $70^{\circ}$
- Two antegrade orifices (major, minor)
- Two to three washing jets  
Medtronic-Hall (diagram below):  
large central + small peripheral jets  
Bjork-Shiley (TEE shown): small  
peripheral jets



# Bioprosthetic Valves

## Bioprosthetic Valves

Carpentier-Edwards (CE)



Photo courtesy of Edwards Lifesciences, Irvine, California

Hancock

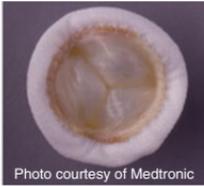
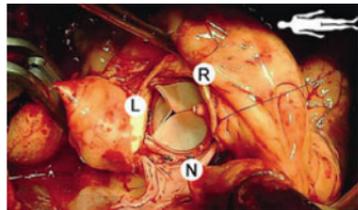
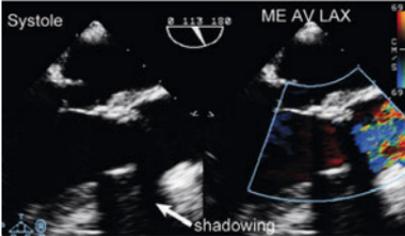
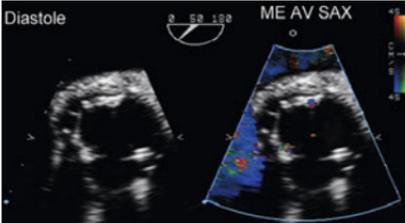


Photo courtesy of Medtronic

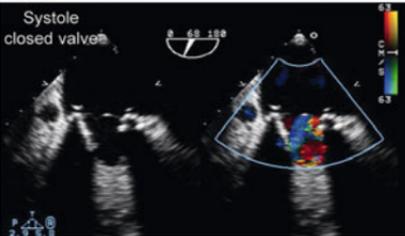
## Stented Valve

- Three stents or struts
- Bovine pericardium (CE) or porcine heterograft (Hancock)
- Three leaflets
- Smaller orifice than stentless valve
- Sized to aortic annulus
- Central gap in pericardial valve



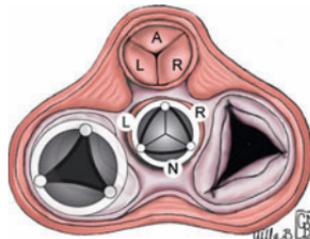
## Aortic Position

- Shadowing from struts in LAX view
- Leaflet mobility (SAX), struts
- Trace valvular AI central, commissural (SAX shown)
- Peak/mean pressure gradients CW Doppler in TG views
- Paravalvular leaks outside sewing ring below the valve in the LVOT

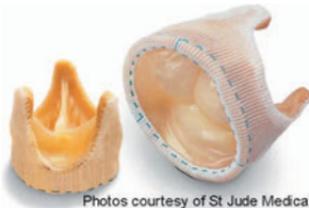


## Mitral Position

- Easily imaged in ME views
- Leaflet mobility
- Trace valvular MR
- Peak/mean pressure gradients
- Paravalvular leaks outside sewing ring in two views
- LVOT obstruction by stent

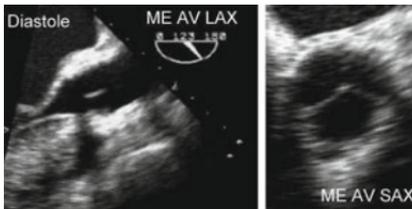


## Patient Prosthetic Mismatch



### Stentless Valve (Toronto SPV)

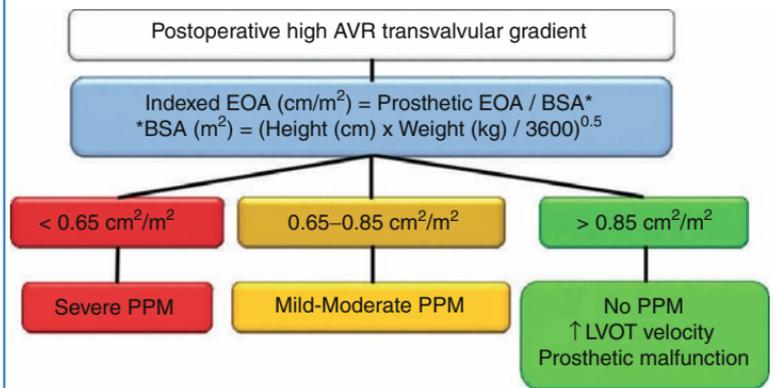
- No stent
- Porcine aortic heterograft
- Three leaflets
- Larger orifice than stented valve
- Only implanted in aortic position
- Sized to sinotubular junction



- $\pm$  acoustic shadowing
- Three leaflets similar to native AV
- Implantation involves valve or valve + root
- Thickened aortic root
- Trace valvular AI
- Small pressure gradient
- Paravalvular leaks

### Patient Prosthetic Mismatch (PPM)

- Prosthesis effective orifice area (EOA) is too small for the patient's size resulting in abnormally high transvalvular pressure gradients.
- May be less relevant in obese patients.
- Well studied with AVR (see below) and can occur with MVR:
  - PPM MVR if  $\leq 1.2$ – $1.3 \text{ cm}^2/\text{m}^2$  occurs in 39–71% of patients.
  - Suspect if persisting pulmonary hypertension.
- When PPM AVR is present, patients have reduced short and long-term survival particularly if there is LV dysfunction.
- Avoidance of PPM in the aortic position may necessitate AVR implantation
  - after patch root enlargement.
  - in the supra-annular position.
  - tilted from the intravalvular position.



# Prosthetic Valve Function

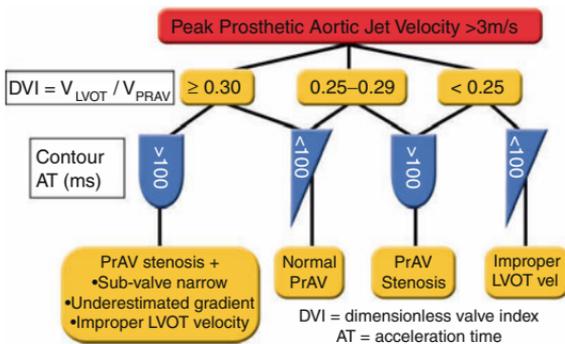
## Aortic Valve Prosthesis

- 2D assess: valve opening and closing
- Doppler:
  - Color: laminar (normal), turbulent, regurgitation (valvular)
  - CW Spectral (see below): flow dependent, avoid being too close to prosthesis
    - Normal: triangular, early peaking, short acceleration time (AT) < 80 ms
    - Obstructed: rounded, mid-peaking, AT > 100 ms, AT/ET > 0.4
  - Flow independent parameters: EOA and DVI (dimensionless valve index)
    - EOA =  $(CSA_{LVOT} \times VTI_{LVOT}) / VTI_{PrAV}$
    - DVI =  $PW\ Velocity_{LVOT} / CW\ Velocity_{PrAV}$
- Associated: LV function, coronary blood flow

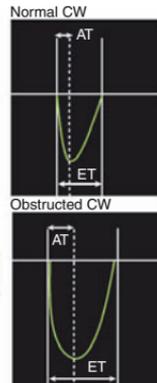
Mechanical and Bioprosthetic AVR Stenosis			
Method	Normal	Possible stenosis	Significant stenosis
Peak velocity (m/s)	< 3	3–4	> 4
Mean gradient (mmHg)	< 20	20–35	> 35
DVI	≥ 0.30	0.29–0.25	< 0.25
EOA (cm <sup>2</sup> )	> 1.2	1.2–0.8	< 0.8
CW through valve	Triangular early peak	Triangular to intermediate	Rounded symmetric
Acceleration time (ms)	< 80	80–100	> 100

Prosthetic Aortic Valve Regurgitation			
Method	Mild	Moderate	Severe
Jet height/ LVOT d <sup>a</sup>	< 25%	25–64%	> 65%
CW density	weak “flat top”	↑ angle on CW	Dense, steep slope
PHT (ms)	> 500	200–500	< 200
PW LV Q: pulmonary Q	Slight ↑	Intermediate	Greatly ↑
Desc. aorta reversal	Early mild	Intermediate	Holodiastolic abd
Regurgitant Volume	< 30cc	30–60cc	> 60cc
Regurgitant Fraction	20–30%	30–50%	> 50%

<sup>a</sup>Nyquist limit 50–60 cm/s, Q = flow



Adapted from Zoghbi et al. J Am Soc Echocard 2009; 22: pg. 990.



## Prosthetic Valve Function

Mechanical and Bioprosthetic MVR Stenosis			
Method	Normal	Possible stenosis	Significant stenosis
Peak velocity (m/s)	< 1.9	1.9–2.5	> 2.5
Mean gradient (mmHg)	≤ 5	6–10	> 10
VTI MV / VTI LVOT	< 2.2	2.2–2.5	> 2.5
EOA (cm <sup>2</sup> )	> 2.0	1.0–2.0	< 1.0
PHT (ms)	< 130	130–200	> 200

- Peak velocity is flow dependent, increasing with: hyperdynamic state, tachycardia, small valve size, valve stenosis, or regurgitation
- VTI is less dependent on HR
- Effective orifice area (EOA): by continuity, bioprosthetic, and tilting disc valves
- Pressure half-time (PHT): dependent on loading conditions and AI; not valid with tachycardia or first degree AV block

Mechanical MVR TTE findings of prosthetic MR with normal PHT			
Method	Normal	Sensitivity	Specificity
Peak E velocity (m/s)	≥ 1.9	90%	89%
VTI PrMV / VTI LVOT	≥ 2.5	89%	91%
Mean gradient (mmHg)	> 5.0	90%	70%
TR jet velocity (>3m/s)	> 3.0	80%	71%
LV stroke volume	> 30%	Moderately	Specific
Flow convergence	Present	Low	Specific

- MR results in hyperdynamic LV with reduced LV systemic output
- CW regurgitation jet with early max velocity
- Paravalvular leak is outside the sewing ring, identify origin, eccentric direction

### Tricuspid Valve Prosthesis

1. 2D assess: valve opening and closing
2. Doppler: inspiratory variation so average over 5 cycles, (\*increased with TR)
  - Peak velocity\*: > 1.7 m/s
  - Mean gradient\*: ≥ 6 mmHg
  - PHT ≥ 230 ms
  - EOA and  $V_{PrAV} / V_{LVOT}$  not validated
3. Associated findings: RV size and function, RA size, IVC size with respiratory variation, hepatic vein flow

### Pulmonic Valve Prosthesis

1. 2D assess: cusp thickening and mobility
2. Doppler findings of stenosis:
  - Color: turbulent antegrade flow
  - Peak velocity / mean gradient
    - Homograft: > 2.5 m/s, > 15 mmHg
    - Bioprosthetic: > 3.2 m/s, > 20 mmHg
  - Elevated RVSP
3. PI assessment similar to native valve (see pg. 106)
  - Color: broad base retrograde jet
  - CW: dense, mid to late peaking, to and fro = sine wave

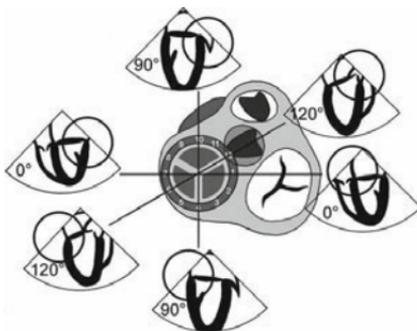
## Prosthetic Valve Function

### Washing (Regurgitant) Jets

- Inside sewing ring
- Short duration
- Depends on prosthesis

### Paravalvular Leaks

- Outside sewing rings
- Longer duration
- Eccentric
- Flow acceleration



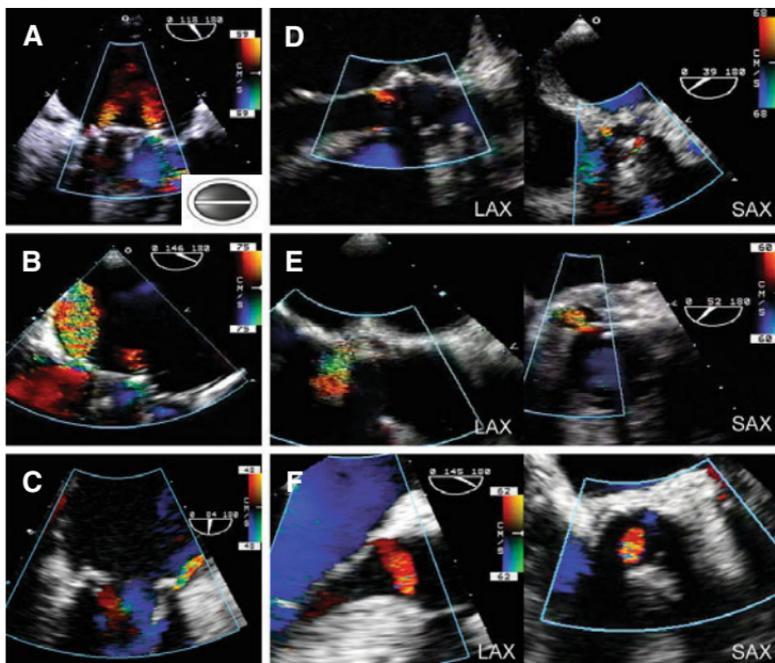
Adapted from

Foster G et al. *Ann Thoracic Surg* 1998; 65:1025.

### Mitral and Aortic Positions

Compare (A) normal washing jets for a bileaflet mechanical MVR with (B, C) paravalvular leaks (systole). Correlate location with the above diagram.

Compare normal washing jets bileaflet mechanical AVR during diastole in (D) ME AV LAX + SAX with (E) posterior and (F) anterior paravalvular leaks. Paravalvular leaks originate outside the sewing ring and are often eccentric.

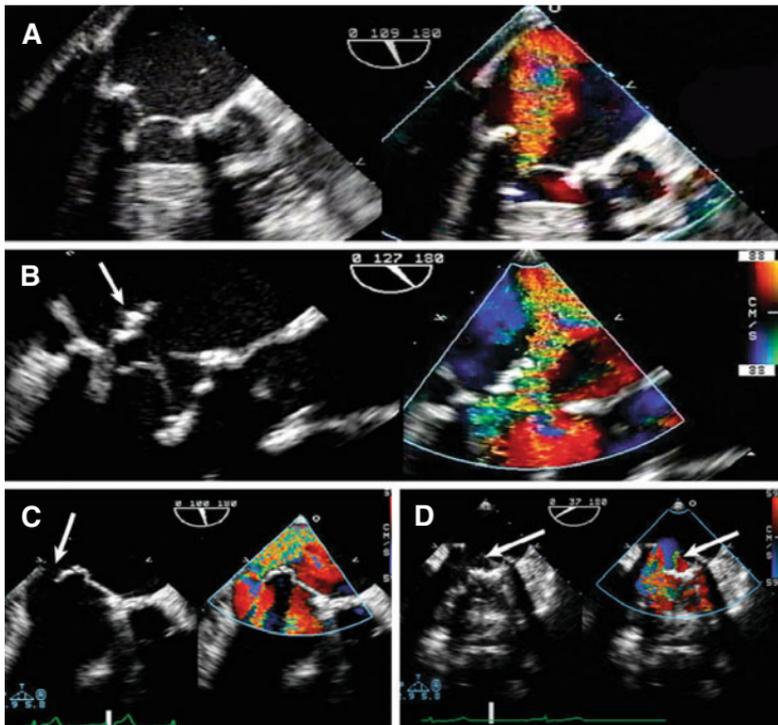


## Prosthetic Valve Function

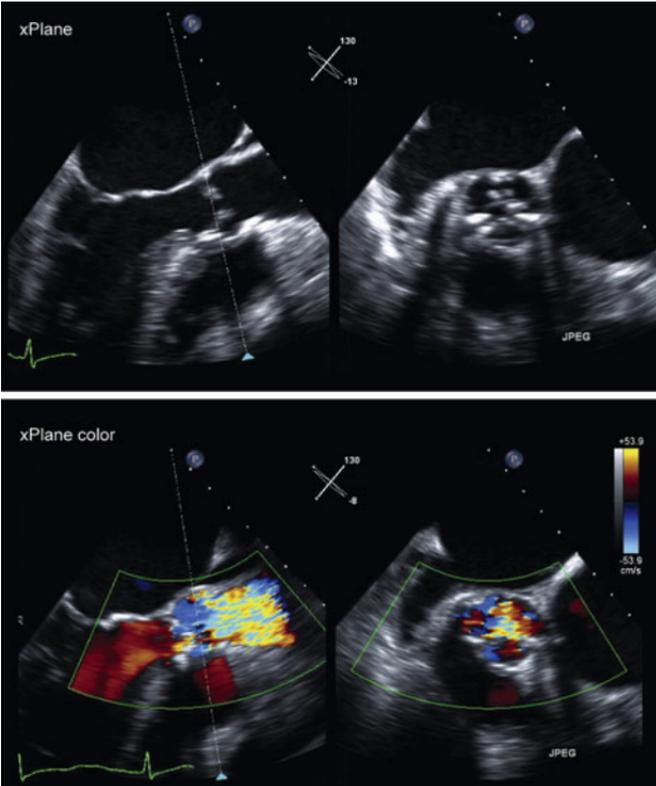
### Prosthetic Valve Dysfunction

Stenosis	Turbulent color, $\uparrow$ Pressure gradient, $\downarrow$ leaflet motion (due to calcification, pannus formation, stuck disc) Calculate valve area, assess PPM (see pg. 123)
Regurgitation	Color, PW Doppler, $\uparrow$ Pressure gradient, valvular vs paravalvular, (due to cusp degeneration, poor disc mobility)
Mass	Thrombus, vegetation
Valve bed	Dehiscence: abnormal rocking motion independent of surrounding structures Pseudoaneurysm: echo free area between aortic annulus and base of AMVL (see pg. 195) Ring abscess: hypoechoic area in adjacent tissue without Doppler communication Fistula: abnormal communication and flow between two sites

- (A) Newly implanted MV bioprosthesis with moderate valvular MR from leaflet suture.  
 (B) Bioprosthetic MV with leaflet vegetation (arrow) and severe valvular MR.  
 (C) Mechanical MVR dehiscence (arrow) with severe paravalvular MR.  
 (D) Mechanical MVR (St Jude) with stuck leaflet (arrow) without diastolic color flow.



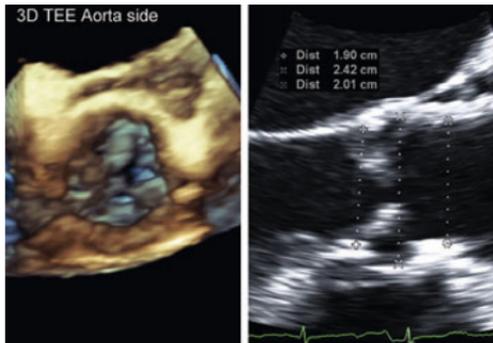
## Transcatheter Valves



Assessment of native aortic valve includes:

- Calcium (2D + 3D)
- Mobility (LAX, SAX)
- Annulus measurement
- Color Doppler

	Annulus (mm)	Valve size
THV	18–22	23
	21–25	26
Core-Valve	20–23	26
	24–27	29



# Transcatheter Valves

There are two commercial systems used for transcatheter aortic valve implantation (TAVI), (A) Edwards Sapien Transcatheter Heart Valve (THV) and (B) Medtronic CoreValve. The TAVI procedure involves placement of a catheter mounted valve delivered either retrograde through a trans-femoral approach or anterograde through a trans-apical approach (only for the THV). The valve is carefully positioned across the native AV, using fluoroscopy and TEE guidance. The externally mounted THV is deployed during inflation of a balloon catheter. The CoreValve is internally contained and self deploys during catheter withdrawal.

Source: Moss RR, et al. JACC Imag 2008; 1: 15-24.



Photo courtesy of Edwards Lifesciences, Irvine, California

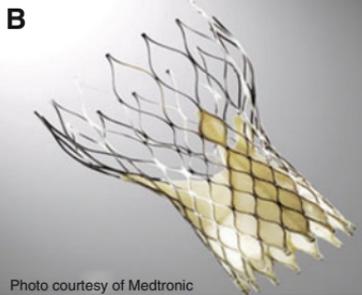
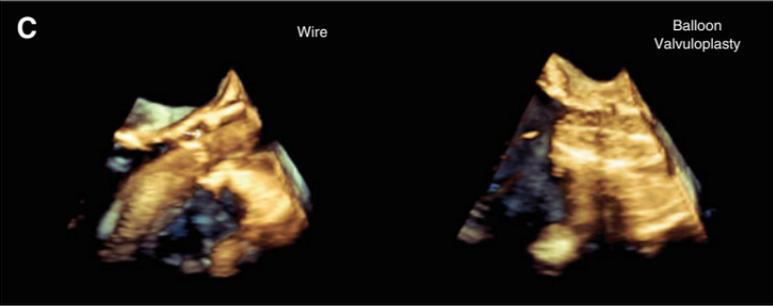
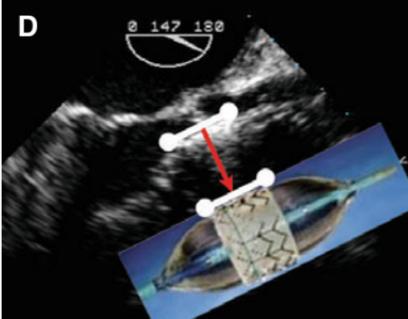


Photo courtesy of Medtronic



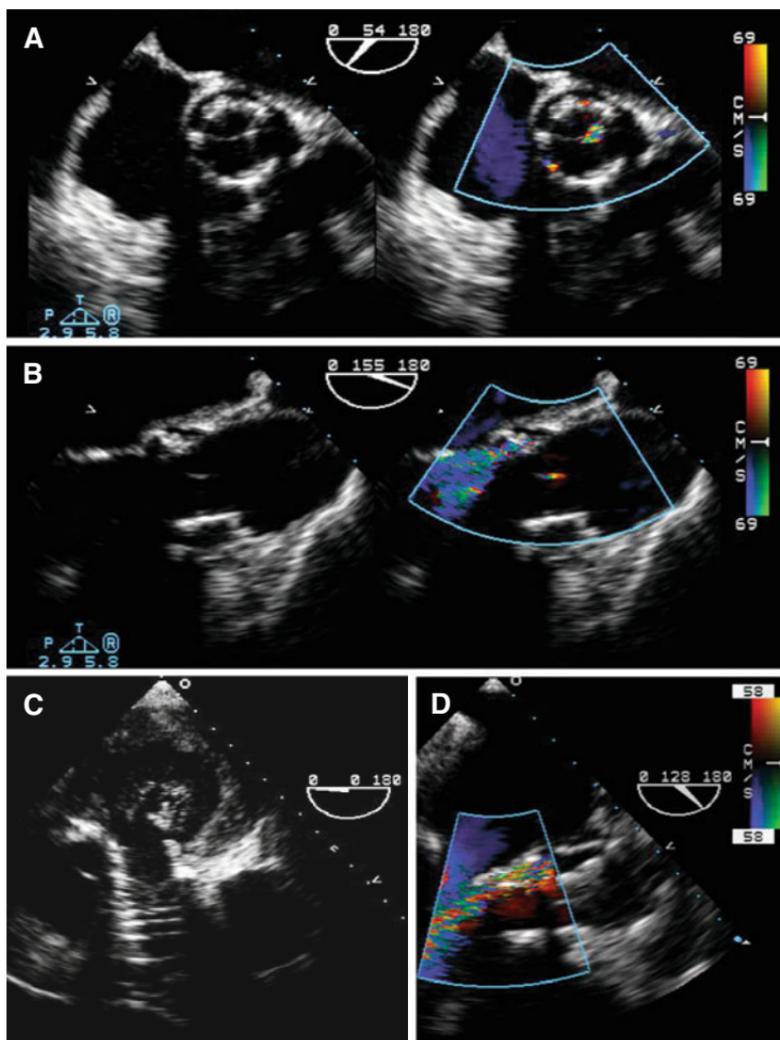
(C) The stenotic native AV is first dilated by balloon valvuloplasty shown in ME AV 3D Live views. (D) The catheter with the undeployed Edwards Sapien THV is positioned with one-half to two-thirds of the valve in the LVOT. During deployment, the THV moves forward slightly so the final prosthetic valve position is at the mid-point of the native aortic valve annulus. The balloon is inflated during a period of rapid ventricular pacing to prevent valve embolization with deployment.



## Transcatheter Valves

Post-valve deployment TEE assessment includes:

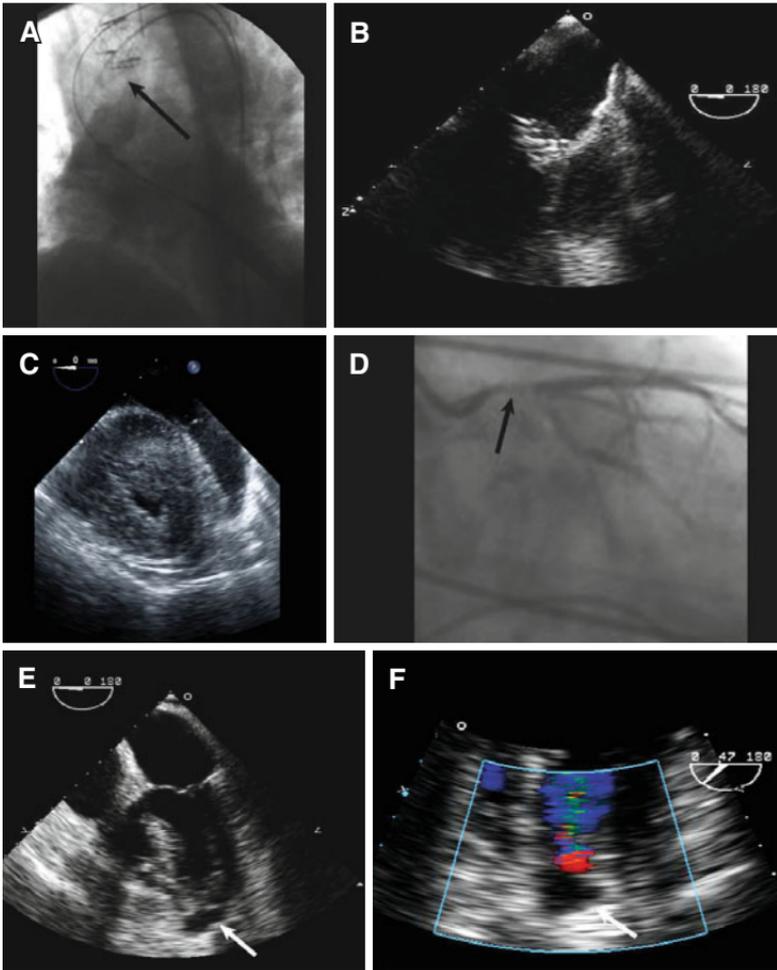
- Confirmation of adequate valve position and stability.
- Presence of paravalvular leak which is common as shown for a Edwards Sapien THV in (A) ME AV SAX and (B) LAX views and the CoreValve in (C) TG and (D) ME AV LAX views. A large paravalvular leak may require further balloon dilatation.
- LV function and coronary flow



## Transcatheter Valves

Rare severe perioperative complications include:

- Systemic valve embolization to mid aortic arch that occurred during valve deployment as seen with (A) fluoroscopy and (B) UE aortic arch LAX view.
- Pericardial effusion and tamponade likely from ventricular perforation during catheter positioning (C) TG mid SAX view.
- Acute left main (LM) coronary occlusion from displaced calcium following balloon valvuloplasty (D) diagnosed with angiography.
- Early postoperative LV apical pseudoaneurysm following a transapical approach in (E) ME 4C and (F) ME 4C color Doppler views.



## Mitral Valve Repairs

### Mitral Valve Repairs

#### Mitral Regurgitation pre-CPB

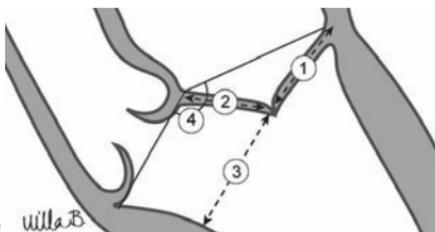
- Location/etiology MR jets
- Severity MR
- Leaflet pathology
- Annulus measurement
- MV inflow spectral Doppler
- Pulmonary vein Doppler
- Ventricular function (R, L)

#### Predictors for Difficult Mitral Repair

- Central MR
- Annular calcification
- Severe annular dilatation
- Bileaflet or multiple segment (> 3)

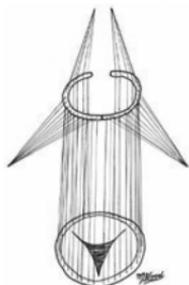
#### Risk of SAM Post-MV Repair

1. PMVL length > 19 mm
2. AMVL/PMVL lengths < 1.3
3. Septal leaflet contact length (SLCL) < 25 mm
4. Mitro-aortic angle  $\leq 130^\circ$



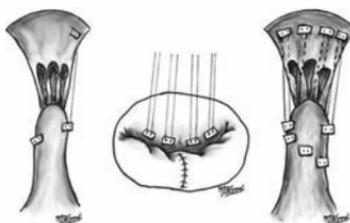
### Annuloplasty

Repair for a dilated posterior annulus when the leaflets fail to coapt. Sutures are placed in the annular tissue and passed through an annuloplasty ring. The sutures are placed closer together in the area of the commissures and the posterior leaflet, which results in a "gathering up" of the posterior annulus. Complete or incomplete, flexible or rigid ring can be used. Image the annular ring near the MV annulus in ME 4C, commissural, 2C, and AV LAX views. The incomplete ring is absent anteriorly in the 4C view, but present in  $60^\circ$ - $120^\circ$  views.



### Artificial Chordae

Repair of chordae, using Gortex to create artificial chordae. Suture is attached to papillary muscle tips, through the mitral leaflet edge, and tied at an approximate length.

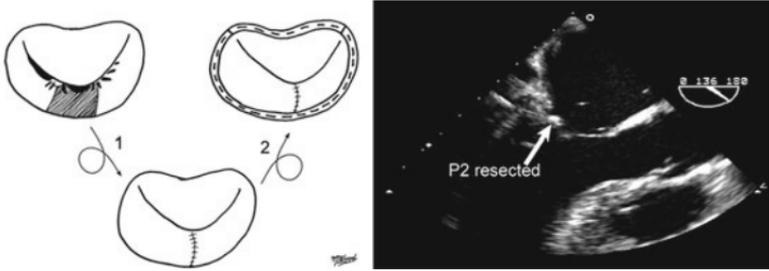


## Mitral Valve Repairs

### Quadrangular Resection (+ Sliding Plasty)

Repair of ruptured chordae to the posterior leaflet. It consists of resecting the ruptured chordae and a leaflet portion, reapproximating the leaflet, and reconstructing the annulus. To support the repair and adapt the annulus to the amount of tissue remaining, a ring annuloplasty (partial or complete) is often performed.

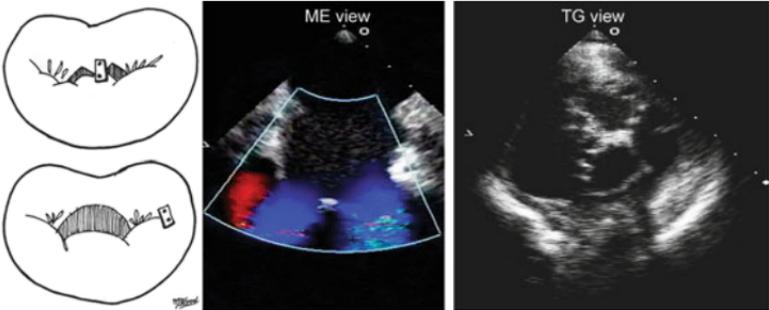
Note short fixed P2 segment of the posterior leaflet (ME 4C, AV LAX views). The large anterior leaflet moves to coapt with the fixed (P2) segment.



### Alfieri Repair

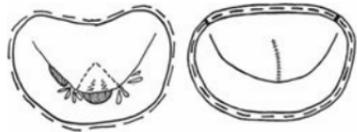
Repair technique for AMVL/dual leaflet prolapse, commissural lesions, PMVL prolapse with severe MAC. Anchors free edge of prolapsing leaflet to corresponding free edge of opposing leaflet (Edge-to-Edge). Repair of prolapse in the leaflet middle portion results in a valve with two openings; prolapse close to a commissure, has a smaller valve opening.

In this example, P2 and A2 segments are sutured together. In the ME 60° view, this is seen as a fixed leaflet, shown here with color Doppler. In the TG MV SAX view, the leaflets form a figure of 8. Planimetry of each orifice will give the MV area.



### Anterior Leaflet Repair

Repair technique for ruptured chordae of the AMVL. A triangular section with the ruptured chordae is resected and the leaflet reapproximated. This may be combined with artificial chordae, if needed, to support the valve mechanism.



# Aortic Valve Repairs

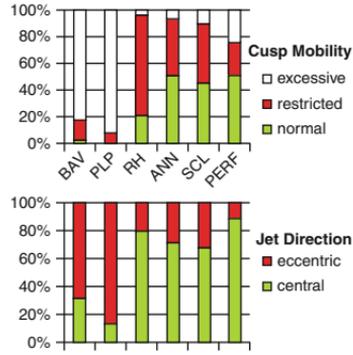
## Aortic Valve Repairs

**El Khoury Classification: AI etiology related to cusp mobility**

Source: El Khoury G. *Curr Opin Cardiol* 2005; 20:115-21.

	Type I (a-d)	Type II	Type III
Cusp motion	Normal, reduced coaptation	Excessive	Restricted
AI direction	Central	Eccentric	Eccentric or central
Etiology	a) Dilated ascending aorta b) Dilated aortic root c) Dilated aortic annulus d) Fenestration	Prolapse Flail	Commissural fusion Calcification

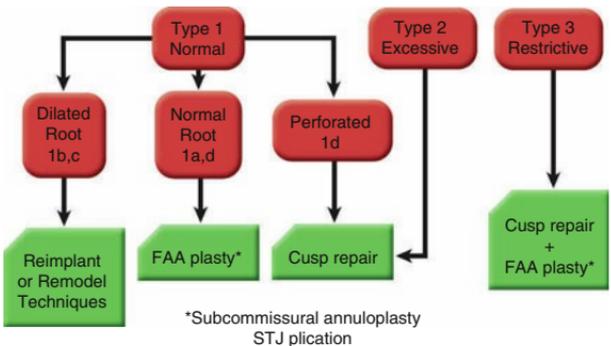
- Aortic Insufficiency Pre-CPB**
- Etiology (cusp vs aortic root)
  - AI direction and severity
  - Measure annulus, sinuses, STJ, Asc aorta
  - LV size, function, SWMA
- Aortic Insufficiency Post-CPB**
- Adequacy of repair (< mild AI)
  - Root morphology restored
  - LV function global/SWMA



BAV (bicuspid), PLP (prolapse), RH (rheumatic heart), ANN (annulus), SCL (sclerosis), PERF (perforation)

Source: Cohen et al. *J Am Soc Echocardiogr* 1996;9:508-15.

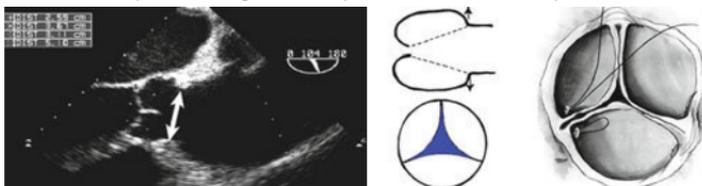
### AV and Root Sparing Surgical Techniques



## Aortic Valve Repairs

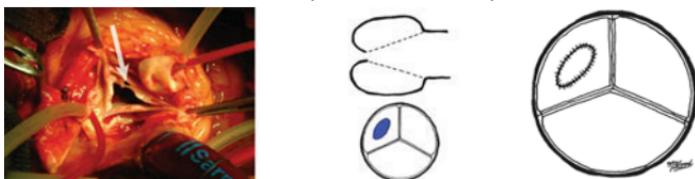
### Annular Dilatation and Plication/Annuloplasty

STJ widens with downward stretching of the commissures. Repair sutures are placed around the commissures (not cusps) reducing the commissural area. This plicates the aortic wall displacing the commissures medially, preserving cusp function. The lower down the suture is placed, the greater the plication and leaflet coaptation area.



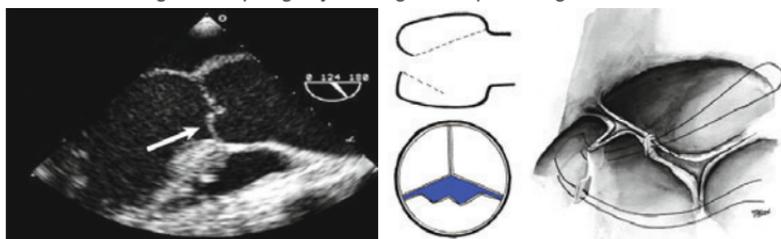
### Cusp Perforation and Patch Closure

Perforations in the cusp causes AI jets that originate at the cusp level. Autologous pericardium is used to oversee and repair holes in the cusp.



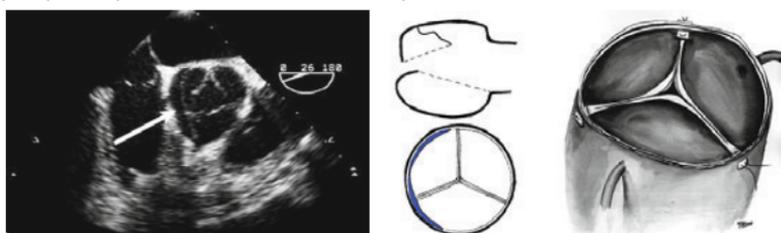
### Cusp Prolapse and Cusp Resuspension

Cusp prolapse results from lack of commissural support or an elongated cusp. Repair shortens the elongated cusp edge by suturing the cusp free edge to the aortic wall.



### Commissural Prolapse and Resuspension

Aortic dissection that extends into the root disrupts the commissure leading to cusp prolapse. Repair involves commissural resuspension.



# Aorta

Anatomy and TEE Views .....	138
Aortic Arch Arteries.....	139
Aortic Atheroma.....	140
Epiaortic Scanning.....	141
Intramural Hematoma.....	142
Aortic Aneurysm .....	143
Valve Sparing Procedures.....	144–145
Sinus of Valsalva Aneurysm .....	146–147
Aortic Dissection .....	148–149

## Anatomy and TEE Views

### Aorta Anatomy

Thoracic aorta is divided into four sections:

1. Aortic root: AV to sinotubular junction (STJ)
2. Ascending aorta: STJ to innominate artery
3. Aortic arch: innominate artery to L subclavian artery
4. Descending aorta: distal to L subclavian artery

Aorta wall has three layers: adventia, media, intima

Size: Ascending aorta length: 7–11 cm

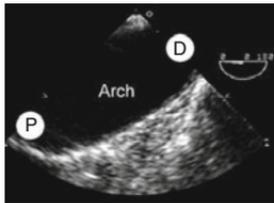
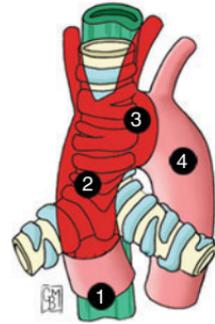
Aorta diameter: 35 mm  $\pm$  2 mm

Wall thickness 1–2 mm

Pathology:

- Dilatation (35–50 mm)
- Aneurysm (> 50 mm)
- Dissection (intimal flap)
- Atheromatous disease (plaque, ulceration, hematoma)

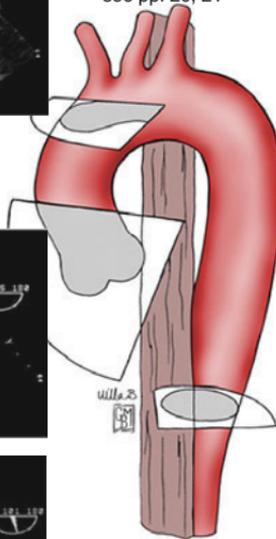
Blind spot: Region of distal ascending aorta and proximal arch obscured by air filled trachea and difficult to image with TEE, shown in red above.



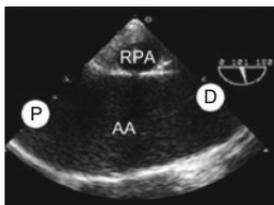
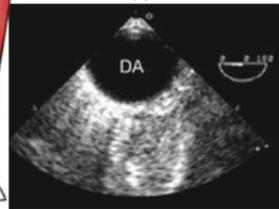
UE Aortic Arch  
SAX (90°)  
LAX (0°)  
see pp. 20, 21



ME Ascending Aorta (AA)  
SAX (0°), LAX(90°)  
see pp. 22,23



Descending Aorta (DA)  
SAX (0°) and LAX (90°)  
see pp. 18,19



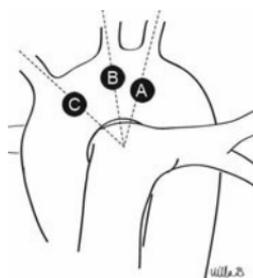
P Proximal  
D Distal



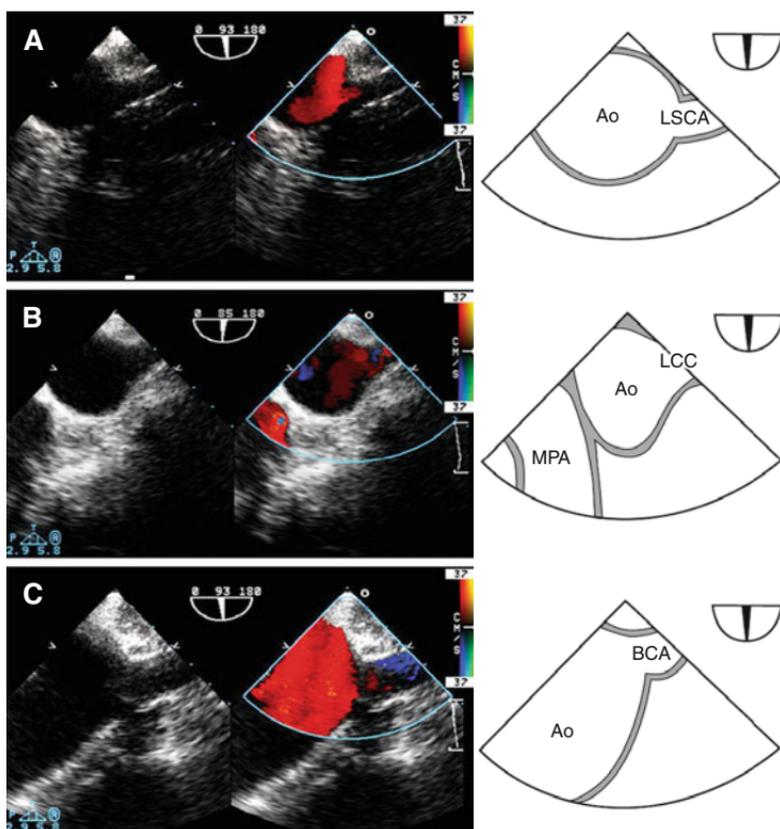
## Aortic Arch Arteries

### Aortic Arch Arteries

Upper esophageal views of the transverse aorta and proximal arch vessels with anatomical correlation. (A–C) Views of the arch vessels are obtained with a left to right rotation of the TEE probe. (A) From an UE Aortic Arch SAX view at 90°, the discrete appearing distal left subclavian artery (LSCA) is imaged. (B) Further rightward probe rotation images the main pulmonary artery (MPA) in LAX and the broader-based origin of the left common carotid artery (LCCA). (C) The most proximal is the innominate (or brachiocephalic) artery (BCA) that gives rise to the right carotid and subclavian arteries. The aortic arch is seen off-axis with the innominate artery in LAX.



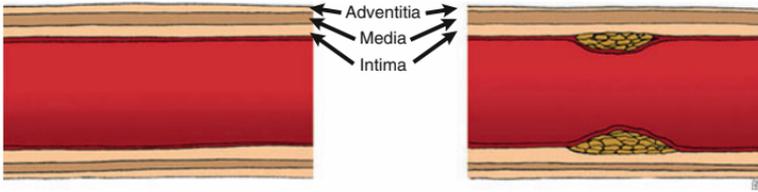
Source: Orihashi K et al. *J Thor Card Surg* 2000; 120:460-72.



# Aortic Atheroma

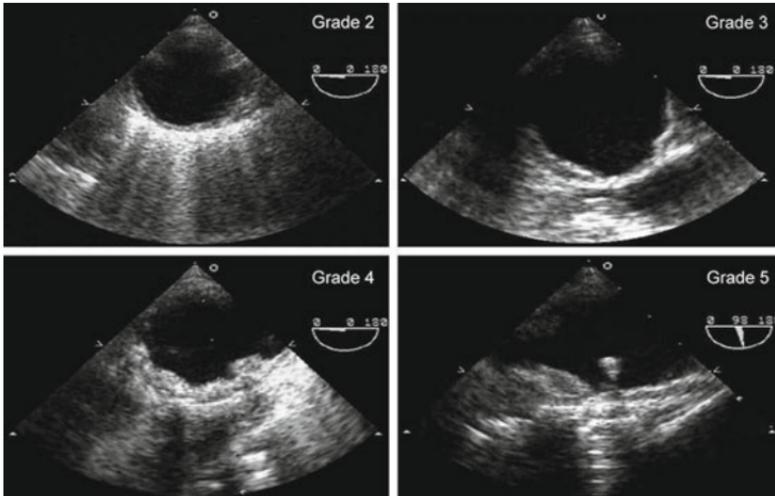
## Aortic Atheroma

- Location: ascending < arch < descending aorta
- Size
- Consistency: thickened intima, irregular, ± calcium
- Ulcerated plaque ± mobile/sessile components
- Atheroma grading: different grading systems based on echo appearance, though none has proven superior to another



## Atheroma Grade (Source: Katz ES, et al. J Am Coll Card 1992; 20:70-77.)

1. Normal aorta
2. Extensive intimal thickening < 3 mm, smooth
3. Protrudes < 5 mm into aortic lumen, irregular, sessile
4. Protrudes > 5 mm into aortic lumen, irregular, sessile (↑ stroke risk)
5. Mobile atheroma of any size (↑ stroke risk)



## What to tell the surgeon

1. Size: Measure thickness and height from intima to adventitia
2. Location of maximal plaque
3. Identify presence of mobile components
4. Atheroma burden: ratio of plaque area/aortic area

## Epiaortic Scanning

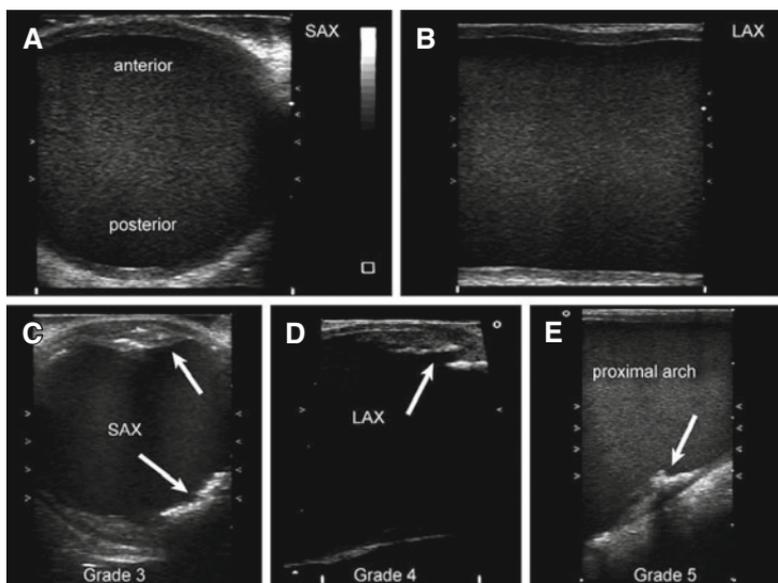
### Epiaortic

Epiaortic scanning uses a high frequency (> 7 MHz) ultrasound probe in a sterile sheath placed by the surgeon directly on the aorta. A linear probe gives a rectangular image. A standard transthoracic probe gives a fan-shaped sector so a stand-off with saline may be preferred to optimize imaging of the anterior aorta. Consider epiaortic scanning if severely calcified descending, arch, or proximal ascending aorta.

Source: Glas K, et al. *J Am Soc Echocardiogr* 2007;11:1227-35.



Epiaortic images of a normal ascending aorta in (A) SAX and (B) LAX obtained using a linear array probe. The image width is the width of the probe, the anterior aortic wall is closest to the probe. (C–E) Aortic atheromatous plaques (arrows) are imaged by epiaortic scan in the ascending aorta and proximal arch. The location, size, and complexity of the lesions can be better identified using this technique.

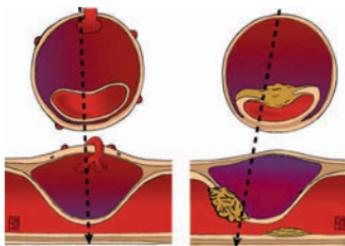


## Intramural Hematoma

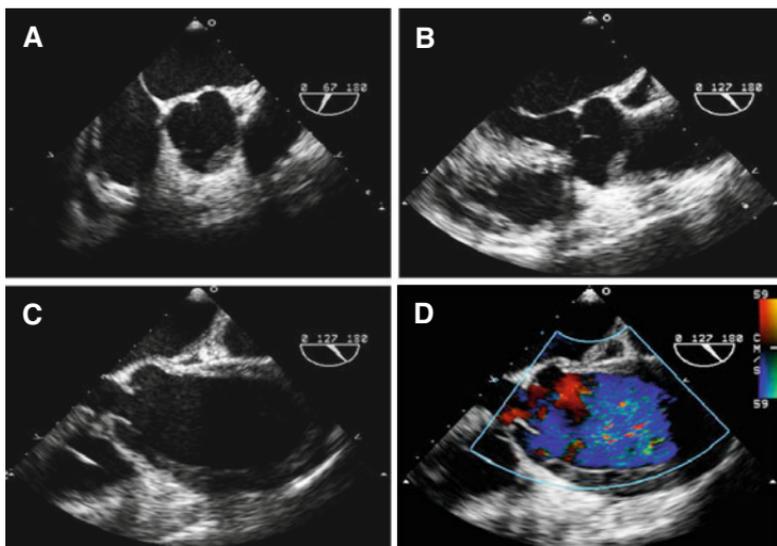
### Intramural Hematoma

Variant of classic aortic dissection  
 Similar classification Type A, Type B  
 Hematoma within aortic wall

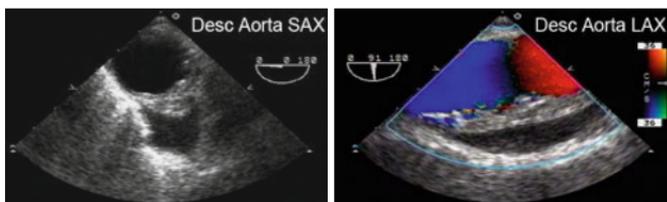
- Aorta thickening > 7 mm  
     inner intima to outer adventitia
- Longitudinal extent 1–20 cm
- Central displaced intimal calcification
- Layered appearance
- Absence of intimal tear
- No flow in hematoma



Aortic hematoma in the aortic root localized near the right sinus of Valsalva in (A) ME AV SAX and (B) ME AV LAX views. More extensive hematoma involving the ascending aorta in (C, D) ME Ascending Aortic LAX views without and with color Doppler.



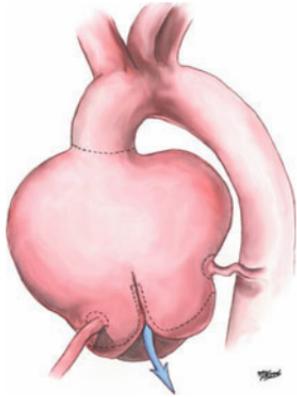
Contained rupture of the descending aorta shows an intimal flap, surrounding hematoma and false aneurysm. In Descending Aortic SAX looks like a left pleural effusion and in LAX the contained space is adjacent to the aorta with no flow by color Doppler.



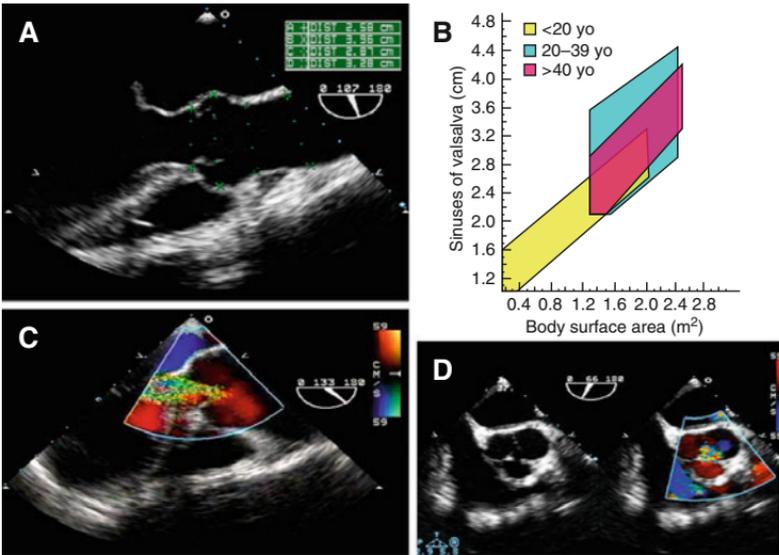
# Aortic Aneurysm

## Aortic Aneurysm

- True aneurysm involves dilatation of all wall layers
- Location: ascending, arch, descending
- Size:  $>1.5 \times$  normal diameter
- Associated findings (AI, thrombus, atheroma)
- Etiology: atherosclerosis, HBP, AS, Marfan's
- Surgery if: sinuses  $> 40$  mm  
     ascending aorta  $> 50$  mm aortopathy  
     ascending aorta  $> 55-60$  mm no aortopathy



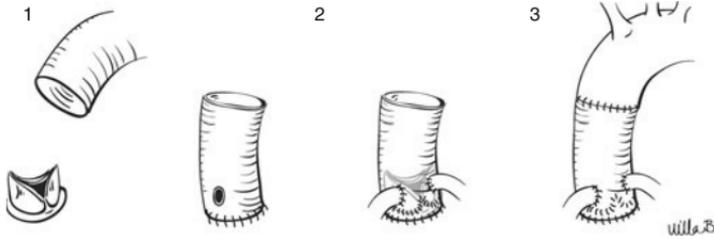
Ascending aortic aneurysm may also have dilatation of aortic annulus, sinuses, STJ, or arch. (A) Measure each site of the aortic root in mid-systole for the extent of pathology. (B) Nomograms for size are indexed to BSA as shown here for the sinuses of Valsalva. (C, D) Poor central AV cusp coaptation causes central AI shown in ME AV LAX and SAX views.



## Valve Sparing Procedures

### Aortic Valve Sparing Procedures

- Aorta is transected above the STJ
  1. Sculpted aortic root is dissected, retaining the commissural pillars
  2. Dacron graft is sutured to heart base using reimplantation or remodeling technique
  3. Coronaries are re-implanted and the ascending aorta is re-anastomosed
- Aortic valve sparing is possible if the cusps are noncalcified, not excessively thinned and sufficiently mobile. Aortic root dimensions are less important.



### Reimplantation Technique (David and Feindel)

Straight Dacron graft is used with suspension of the native commissural pillars (A–C)

Advantages: hemostatic, annulus stable, reproducible procedure

Disadvantages: Three suture lines, lack of sinuses

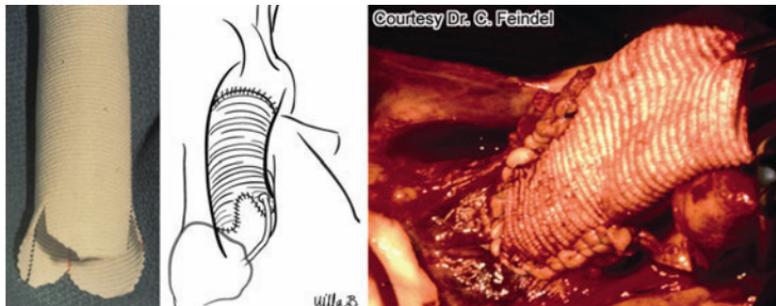


### Remodeling Technique (Yacoub, David II)

Sculpted Dacron graft is sutured to native commissural pillars

Advantages: Two suture lines, neo-sinuses

Disadvantages: no annulus support, difficult to reproduce

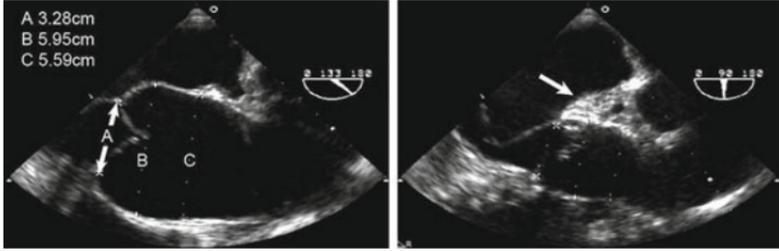


## Valve Sparing Procedures

### Annuloaortic Ectasia (often in Marfan's syndrome)

Pre: dilatation of aorto-ventricular junction (annulus), may involve sinuses, STJ (no waist) and ascending aorta, thinned aortic cusps with reduced coaptation and AI.

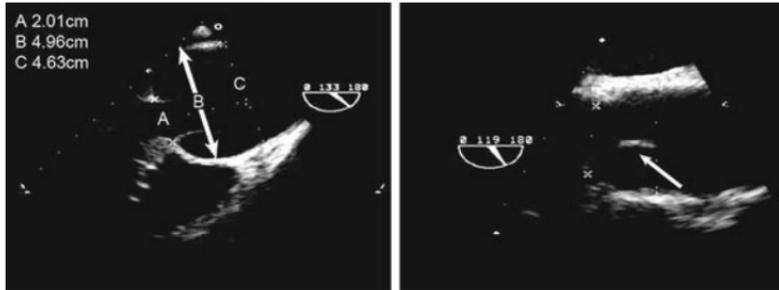
Post-valve sparing: note thickened aortic root from patch annuloplasty of aortic annulus (arrow) and lack of sinuses and tapered STJ.



### Aortic Root Aneurysm

Pre: normal annulus size, dilated sinuses of Valsalva and STJ (no waist), reduced cusp coaptation with AI

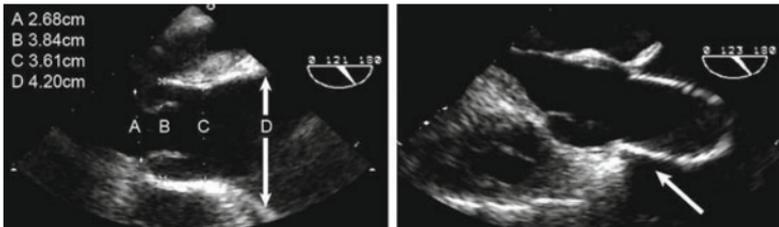
Post-valve sparing: Zoom of the aortic root shows coaptation above annular plane, thin root without patch annuloplasty, measured coaptation length  $\geq 7$  mm (arrow).



### Ascending Aortic Aneurysm (HBP, aortic stenosis)

Pre: normal size annulus and sinuses of Valsalva, dilatation occurs after STJ in the ascending aorta, good cusp coaptation without AI.

Post-valve sparing: Dacron graft visible distal to STJ (arrow). The "ball shape" of the aortic root from the sinuses of Valsalva is retained.



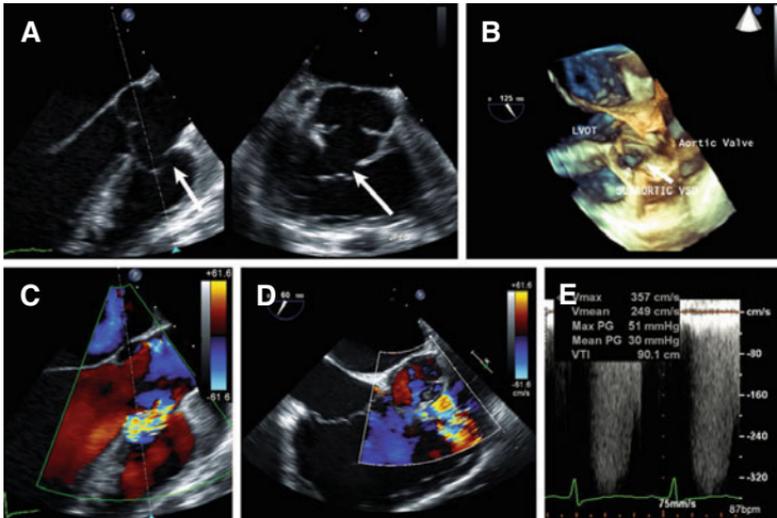
# Sinus of Valsalva Aneurysm

## Sinus of Valsalva Aneurysm

- Etiology: congenital, acquired, male (4) : female (1)
- Aortic wall weakness with fusiform (true aneurysm) or focal “windsock” deformity
- Congenital: single sinus, Acquired: diffuse 2° to Marfan’s, syphilis, trauma
- Location: right (65–85%), non (10–30%), left (< 5%)
- Associated findings: VSD, bicuspid AV, AI, pulmonic stenosis, coarctation, ASD
- Complications: rupture (RA > RV > LV > PA/IVS), endocarditis, thrombus, MI
- 2D imaging:
  - Sinus dilatation: single (congenital) or diffuse (acquired)
  - Location + size of defect
  - Cardiac chamber penetrated
  - Windsock deformity of the sinus
  - Thrombus in sinus
  - RV/LV volume overload /dilatation, systolic function
- Doppler findings:
  - Color: flow into aneurysm
    - Location of rupture, echo dropout at windsock tip
    - Shunt direction
  - Spectral: Peak/mean pressure gradients across intracardiac fistulae
    - Aortic-intracardiac fistula continuous (S+D) high velocity unidirectional flow
    - VSD demonstrate high velocity systolic flow + low velocity diastolic flow

## Right Sinus of Valsalva Aneurysm (SOVA)

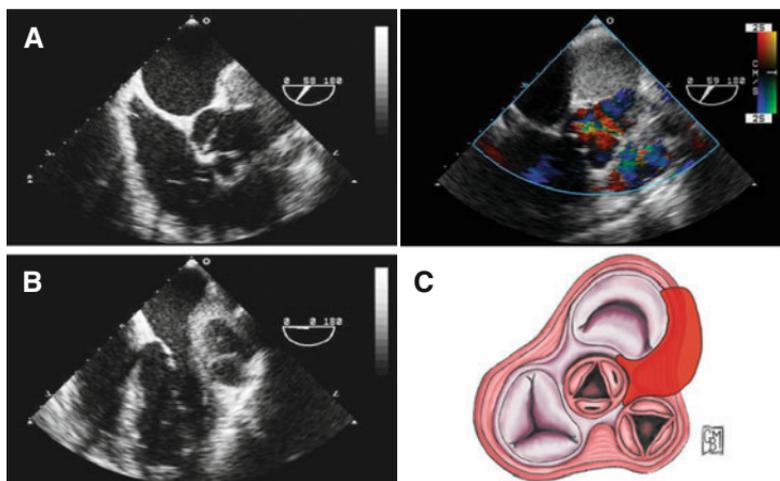
Right SOVA (arrow) is seen in (A) 2D ME AV LAX and SAX views with the windsock orifice in (B) 3D AV LAX view from the aorta. Color Doppler does not show AI in the (C) AV LAX view, but flow from the aorta into the RV is shown in the (D) RVOT view. (E) CW Doppler demonstrates a peak gradient of 51 mmHg.



## Sinus of Valsalva Aneurysm

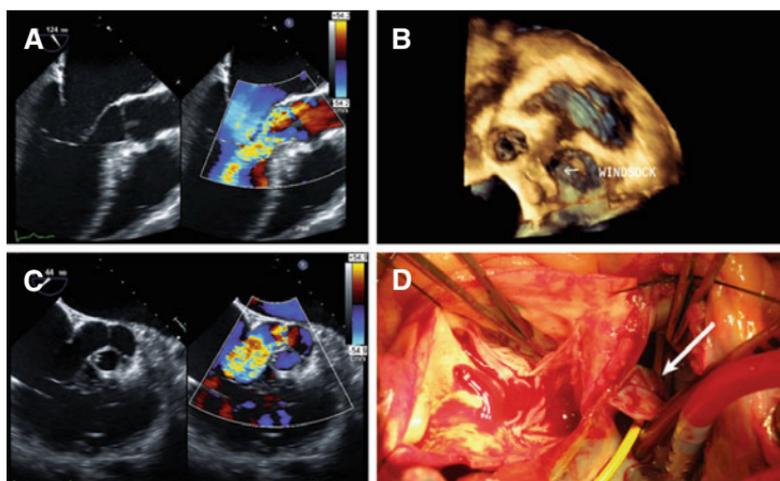
### Left Sinus of Valsalva Aneurysm (SOVA)

(A) Ruptured large left SOVA is seen contained by pericardium in the ME AV SAX views with thrombus and minimal flow by color Doppler (low Nyquist). (B) ME 4C view the thrombus is seen extending to the lateral MV annulus. (C) Diagram of ruptured SOVA thrombus as seen in relation to the base of the heart.



### Noncoronary Sinus of Valsalva Aneurysm (SOVA)

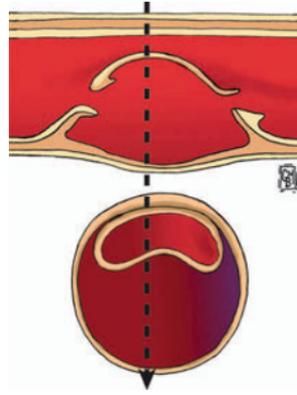
Pathology is not obvious in (A) ME AV LAX, but present in (C) ME RVOT view with AI. Compare (B) 3D full volume of the SOVA with the (D) intraoperative findings.



# Aortic Dissection

## Aortic Dissection

- Tear in intima, blood in media creates a false lumen with blood flow
- Identify intimal flap
  - Discrete sharp edge
  - Seen in two separate views
  - Oscillating, undulating movement
  - Interrupts color flow
  - Not outside lumen or across anatomic planes
- Location of entry and exit sites (color Doppler)
  - STJ, left subclavian artery
- Extent of dissection (distal to proximal)
- True vs false lumens (see below)
- Stanford classification (see next page)
  - Type A: ascending aorta
  - Type B: descending aorta
- Complications:
  - Aortic insufficiency (50–70%) quantify, mechanism
  - Coronary dissection (10–20%): flap flow
  - Pericardial effusion, pleural effusion
  - LV function: global, SWMA



Source: Evangelista A, et al. Eur J Echocardiogr 2010;11(8):645-58.

**True lumen (TL)**  
 Smaller lumen  
 Expands in systole (M-mode)  
 Color prominent  
 No smoke

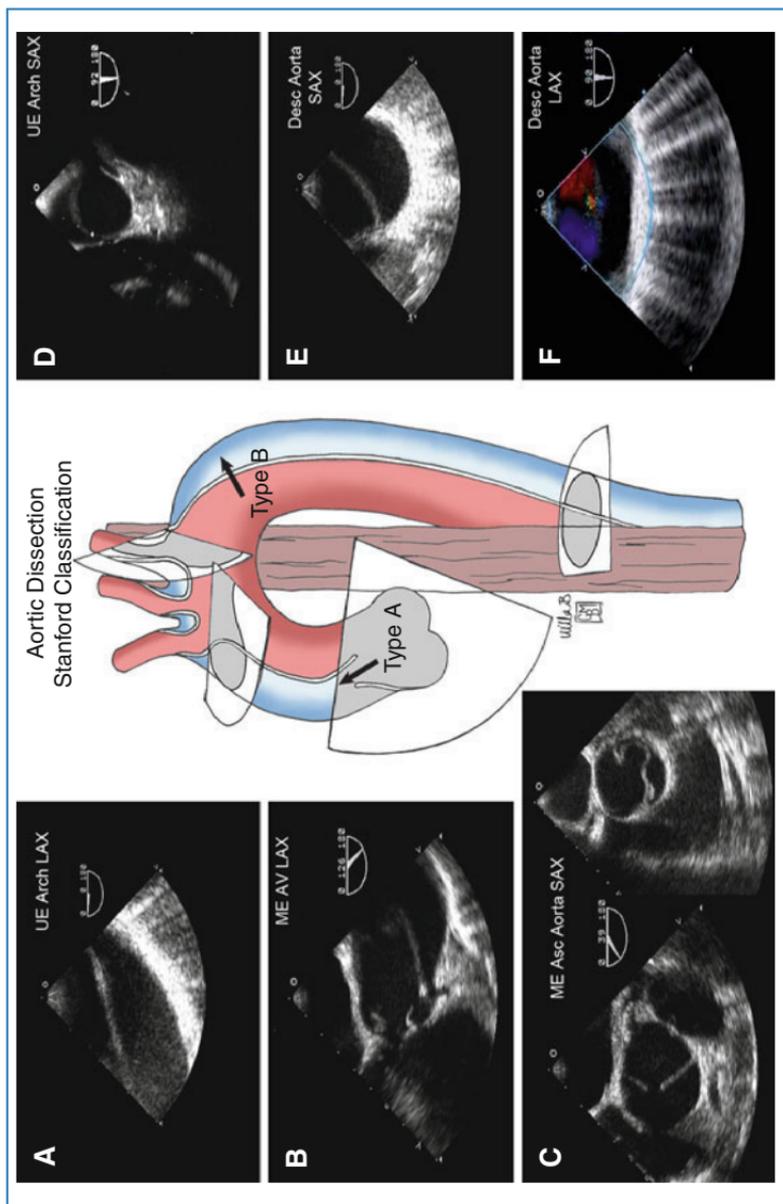
**False lumen (FL)**  
 Larger lumen  
 Expands diastole  
 Color less prominent  
 Clot/smoke present

Test modality	Sensitivity (%)	Specificity (%)
TTE	50–80	60–96
TEE	97–100	100
CT	67–100	80–100
MRI	98–100	87–100

### What to tell the surgeon

- Site origin and extent of intimal flap
- Flow in false lumen
- AI, pericardial effusion, pleural effusion
- LV function (global vs. segmental)
- Aortic root dimensions, AV structure

## Aortic Dissection



# Congenital Heart Disease

Overview and Classification.....	152–154
Inter-atrial Septum and PFO .....	155
Atrial Septal Defects .....	156–159
Ventricular Septal Defect.....	160–163
Tetralogy of Fallot .....	164–165
D-Transposition of the Great Arteries .....	166–167
Mustard Procedure .....	168–169
Fontan Procedure.....	170–171
Ebstein's Anomaly .....	172–173
L-Transposition of the Great Arteries.....	174
Cor Triatriatum.....	175
Patent Ductus Arteriosum .....	176
Subaortic Membrane.....	177

## Overview and Classification

### TEE Segmental Approach

- Determine cardiac sidedness (situs) depends on atrial mass
  - Situs (arrangement): solitus (usual), inversus (mirror image), ambiguous (R or L)
  - Abdominal situs: solitus (usual), inversus, heterotaxia
- Determine cardiac position
  - Based on position in the thorax (dextro/meso/levo-position)
  - Based on cardiac apex orientation (dextro/meso/levo-cardia)
- Identify three segments
  - Atrial segments: differentiated by atrial appendage R/L appearance
  - Ventricular segments: differentiate described below
  - Arterial segments: PA bifurcated into LPA/RPA, Aorta: coronaries originate

Right atrium morphology		Left atrium morphology	
Wide-necked appendage Extensive pectinate muscles Valves of IVC and coronary sinus		Narrow-necked hook-like appendage Smooth walled except for appendage	
	RV	LV	
Atrioventricular valve	Trileaflet	Bileaflet (unless AMVL cleft)	
Leaflet attachment	Septum	No septal	
Annulus location	More apical	More basal	
Apex	Prominent	Less prominent trabeculations	
Moderator band	Present	Absent	
Infundibulum	Present	Absent	
Ventricular size, shape, and wall thickness do not distinguish R and L Morphologically indeterminate if coarse trabeculations and no interventricular septum (univentricular heart) Tricuspid valve always attaches to RV; mitral valve always to LV			

- Define the connections

#### Atrioventricular connection

- Concordant: RA to RV, LA to LV
- Discordant: RA to LV, LA to RV
- Ambiguous: isomeric
- Double inlet (univentricular) connections three possibilities: absent R connection, absent L connection, indeterminate
- Atrioventricular valve morphology: straddling, over-riding, stenotic, regurgitant, dysplastic, imperforate

#### Ventriculo-arterial connection

Two arterial trunks:

- Concordant: RV to PA, LV to Aorta
- Discordant: RV to Aorta, LV to PA
- Valve morphology:
  - Aortic valve always attaches to aorta
  - Pulmonic valve always attaches to PA
- Double outlet: 1 arterial trunk + >½ other connected to same ventricle

One arterial trunk:

- Single outlet: truncus arteriosus IV, truncus type I-III
- Outflow tract: muscular (RVOT), fibrous (LVOT)

# Overview and Classification

## Segmental Approach Congenital Heart Disease

1. Determine Cardiac Sidedness (Situs)

Based on position of morphological RA

**Situs Solitus**  
RA lies to right of LA



**Situs Inversus**  
RA lies to left of LA



**Situs Ambiguus**  
Indeterminant/Isomersim  
Paired mirror image sets of normally single nonidentical organs

**Right**

- R Bronchi (x2)
- R Atria (x2)
- No spleen



**Left**

- L Bronchi (x2)
- L Atria (x2)
- Polyspleen



**Abdominal Situs**  
Position of major unpaired organs

**Solitus**



**Inversus**



**Heterotaxia**



2. Determine Cardiac Position

**Cardiac Position**  
Based on position in thorax

**Dextroposition**



**Mesoposition**



**Levoposition**



**Cardiac Orientation**  
Base to apex axis

**Dextrocardia**



**Mesocardia**



**Levocardia**



3. Identify 3 Segments

**Atrial Segment**

**Right**

- Triangular RAA
- Broad-based RAA
- Terminal crest
- Pectinate muscles (SVC/IVC)



**Left**

- Narrow LAA
- Hook shaped
- No terminal crest



**Ventricular Segment**

**TV/RV**

- Apical SLTV\*
- SLTV\* chords IVS
- Coarse trabecula
- Moderator band
- Supraventricular crest
- \*Septal leaflet tricuspid valve



**MV/LV**

- Fibrous continuity
- No chords to IVS



**Arterial Segment**

**Pulmonary Trunk**

- Bifurcation to RPA and LPA



**Aorta**

- Coronary arteries
- Branches to head



4. Define the Connections

**Veno-Atrial**

- NC/SVC
- Pulmonary veins

**Atrio-Ventricular**

**Concordant**

- RA → RV
- LA → LV



**Discordant**

- RA → LV
- LA → RV



Mirror Image

**Ventriculo-Arterial**

**Concordant**

- RV → PA
- LV → Aorta



**Discordant**

- RV → Aorta
- LV → PA



**Double Inlet Ventricle**  
Connection of both AV valves to predominantly one ventricle

Predominant RV  
Absent LV



Indeterminate



Predominant LV  
Absent RV



**Double Outlet Ventricle**  
Both great arteries arise from predominantly one ventricle



All images courtesy of Willa Bradshaw

153

## Overview and Classification

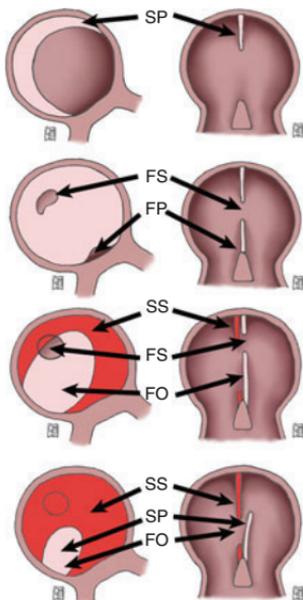
### Congenital Heart Disease Classification

1. Septal defects
  - Atrial septal defects (ASD)
    - Secundum, primum, sinus venosus, and coronary sinus
  - Ventricular septal defects (VSD)
    - Outlet, muscular, inlet, and perimembranous
  - Atrioventricular septal defects (AV canal defects)
2. Disorders of mitral valve inflow
  - Anomalous pulmonary venous drainage (total-TAPVD, partial-PAPVD)
  - Cor triatriatum
  - Mitral Stenosis: supralvalvular, parachute
  - Mitral Atresia
3. Diseases of left ventricular outflow tract (LVOT)
  - Subaortic, supralvalvular stenosis
  - Valvular stenosis
  - Sinus of Valsalva aneurysm
4. Diseases of aorta
  - Patent ductus arteriosus (PDA)
  - Coarctation of the aorta, aortic atresia
  - Truncus arteriosus
  - Vascular anomalies
5. Diseases of tricuspid valve
  - Ebstein's anomaly
  - Tricuspid atresia
6. Diseases of right ventricular outflow tract (RVOT)
  - Subvalvular: tetralogy of fallot (TOF)
  - Valvular: stenosis, pulmonic atresia
7. Chambers and valves are in **abnormal** sequence
  - Atrioventricular discordance (corrected transposition)
  - Ventriculo-great arterial discordance (transposition of great vessels)
  - Double-inlet ventricle (with univentricular heart)
  - Double-outlet right and left ventricles

Source: Russell IA, et al. *Anesth Analg* 2006; 102: 694-723.

Acyanotic	Cyanotic
VSD	D-TGA
ASD	TAPVD
PDA	Truncus Arteriosus
Pulmonic Stenosis	TOF
Coarctation	Tricuspid Atresia
Ebstein's Anomaly	Univentricle

## Inter-atrial Septum and PFO

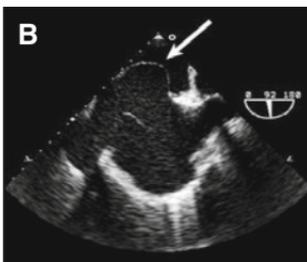
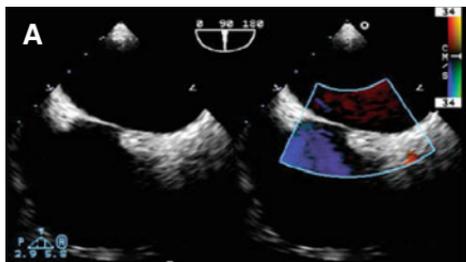


### Embryology of the Inter-atrial Septum (IAS)

- Formation begins with the septum primum (SP) growing down from the dorsocranial wall of the atria towards the endocardial cushions. Above the endocardial cushions a space, the foramen primum (FP) remains.
- Perforations appear in the upper SP and form the foramen secundum (FS), allows for partial reabsorption of the SP.
- The septum secundum (SS) grows from the ventrocranial wall and covers the FS and FP. But leaves an opening the foramen ovale (FO) which is covered by the septum primum (SP).
- The upper septum disappears and the lower portion becomes the valve of the foramen ovale.

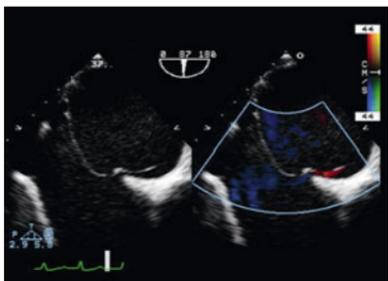
### Normal variants of the IAS

- Lipomatous hypertrophy is a benign fatty infiltration of the surrounding tissue of the IAS with sparing of the thin membranous fossa ovalis.
- IAS aneurysm (arrow) is defined by a mobile IAS with excursions of > 10 mm. These mobile IAS are associated with PFOs and increased risk of stroke. A prominent eustachian valve is also seen.



### Patent Foramen Ovale (PFO)

- Tissue flap in IAS, no tissue deficiency
- Bicaval or AV SAX views look for small gap (flap) in IAS
- Confirm with color Doppler
- Saline contrast (SC) study
- Incidence:
  - 25% autopsy
  - + 5–10% TEE color
  - + 5% SC at rest
  - + 25% SC cough, Valsalva



## Atrial Septal Defects

### Atrial Septal Defects

- Secundum (70%): within fossa ovalis, associated mitral valve prolapse (MVP)
- Primum (20%): inferior septal area, associated  $\pm$  atrioventricular valve abnormalities (cleft MV),  $\pm$  endocardial cushion (inlet VSD), aneurysmal IVS
- Sinus venosus (8%): posterior septal area, SVC (superior) or IVC (inferior) type, associated partial anomalous pulmonary venous drainage (PAPVD) from right lung
- Coronary sinus (2%): inferior septal area close to coronary sinus, unroofed coronary sinus drains into LA which also communicates with RA

### 2D Imaging

- 2D views (ME 4C, RVOT, bicaval)
- Type, location, size of defect
- Volume overload proportional to defect size, results in dilated right side:
  - RA
  - RV, RVH if  $\uparrow$  PAP, paradoxical motion and flattening of the IVS
  - Pulmonary artery has increased flow
- Associated lesions: primum (cleft MV), secundum (MV prolapse), sinus venosus (PAPVD)
- Agitated saline contrast (bubble) study is sensitive in diagnosing
- Atrial septal aneurysms may have a shunt
- PFO in 25% of patients, some only shunt R to L post-Valsalva maneuver
- Device closure up to 38 mm and need rim of surrounding tissue

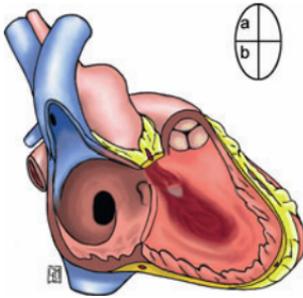
### Doppler

- Color: laminar vs. turbulent flow, reduce Nyquist limit  $< 30$  cm/s
  - Direction of shunt (usually L $\rightarrow$ R)
- PW continuous flow (see next page)
- TR is often present (TV annulus dilatation)
  - Estimate RVSP (pulmonary hypertension)
- PI if dilated PA, turbulent flow in PA due to  $\uparrow$  flow
- MR if cleft mitral valve leaflet
- Identify drainage of all four pulmonary veins into LA
- Qp/Qs shunt ratios: SV Qp and SV Qs sites (see pg. 49)
  - ASD: Qp is PA / Qs is aortic or mitral valve
  - Hemodynamically significant shunt  $> 1.5:1$

#### What to tell the surgeon

- Defect type
- Single or multiple defects
- Size of defect
- Identify all four pulmonary veins
- RV size and function
- PA size
- RVSP from TR jet
- Agitated saline contrast (bubble) study
- Primum ASD look for cleft MV
- Sinus Venosus ASD look for PAPVD
- Persistent shunt post-repair

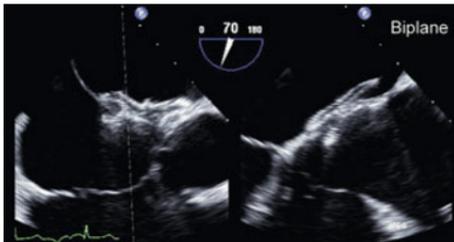
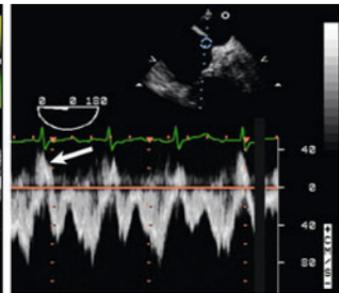
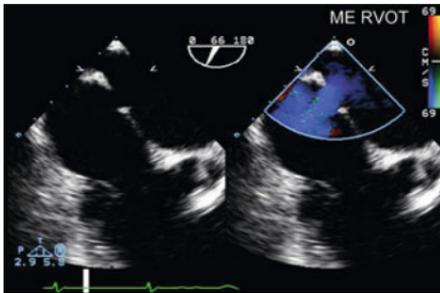
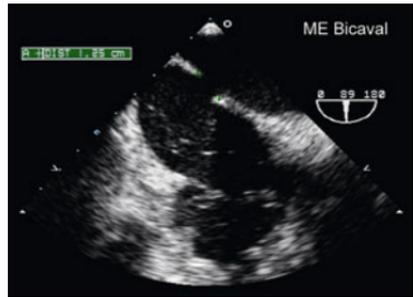
## Atrial Septal Defects



### ASD Secundum

- Most common type of ASD (80%)
- Within the fossa ovalis (center of IAS)
- Bound on all sides by tissue
  - (a) Major axis (bicaval)
  - (b) Minor axis (RVOT)
- May be isolated or part of complex congenital problems
- Associated mitral valve prolapse, PAPVD right pulmonary veins

- ME 4C, AV SAX, RVOT, Bicaval views
- IAS gap (measure size, oval shape)
- Color Doppler:
  - Laminar flow (large nonrestrictive)
  - Turbulent flow (small restrictive)
- PW Doppler:
  - Direction of flow, biphasic
    - L→R in midsystolic + diastolic
    - Flow reversal early systole (arrow) may worsen R→L shunt with IPPV
  - Low velocity < 1.5 m/s
  - Peak velocity inverse relation to size



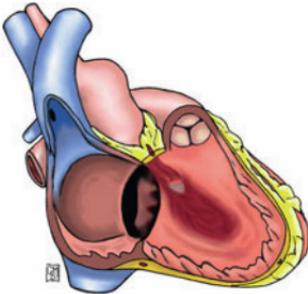
### Device Closure

Intravascular deployed umbrella device across IAS closing ASD secundum.

Complications:

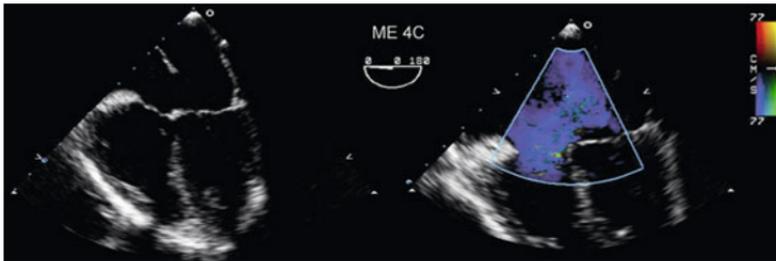
- Clot, thrombus
- Device instability
- Erosion (Aorta → LA fistula)
- Residual shunt

## Atrial Septal Defects



### Primum ASD

- Second commonest type of ASD (20%)
- Located in the lower portion of the atrial septum, involves the atrioventricular septum
- Atrioventricular valves in same plane
- Form of endocardial cushion defect:
  - Partial: primum ASD
  - Complete: primum ASD + inlet VSD + common atrioventricular valve
- Associated defects: cleft MV, subaortic stenosis, double orifice MV, coarctation, PDA, TOF



### 2D Image (ME 4C view)

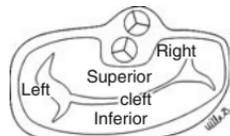
- Absence of IAS above atrioventricular valves
- Both atrioventricular valves (MV, TV) in the same plane
- Measure largest gap with and without color
- RA, RV, PA dilated

### Color Doppler

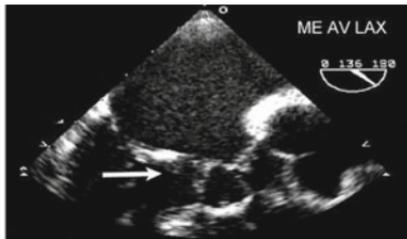
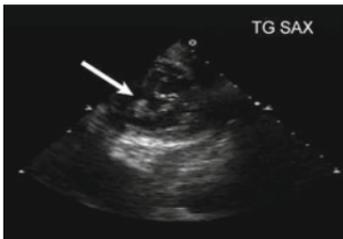
- Color turbulent or laminar (unrestricted) flow, usually L → R, through defect
- Atrioventricular valve regurgitation: systemic MV → MR, venous TV → TR

### Cleft Mitral Valve

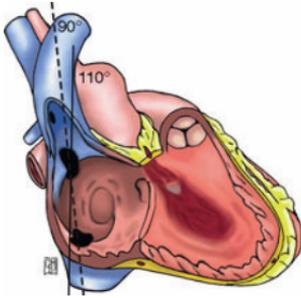
- The cleft is the apposition line between the septal attachments of the superior and inferior bridging leaflets
- Slit-like gap in "anterior leaflet" (TG SAX view arrow)
- Abnormal chordae to base of IVS (ME AV LAX view)
- Eccentric MR originates at the cleft



Common atrioventricular valve



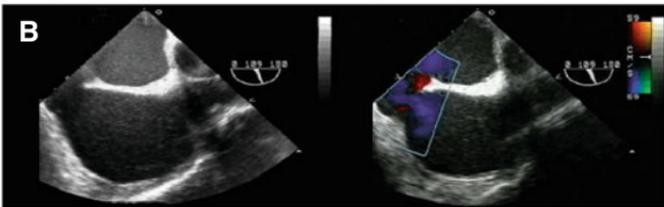
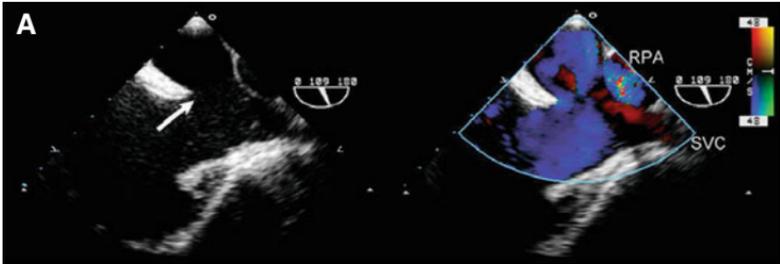
## Atrial Septal Defects



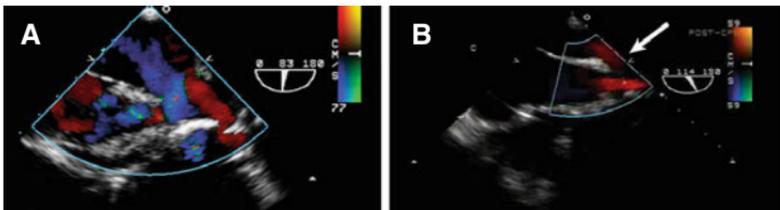
### Sinus Venosus ASD

- Less common type of ASD (8%)
- Located posterior in the upper (SVC) or lower (IVC) portion of the atrial septum
- Defect in the common wall of the vena cava, RA, and pulmonary veins
- Associated partial anomalous pulmonary venous drainage (PAPVD) usually of the right lung
  - SVC type: RUPV, RLPV
  - IVC type: RLPV

Modified bicaval view (109°) shows a discontinuity in the vena cava. (A) **SVC type** occurs between LA, SVC, and RA with the RPA in view. (B) Advancing the probe towards the liver images the **IVC type**. Measure defect size (arrow). Color Doppler shows laminar blue flow from an unrestricted L→R shunt in both these cases.



**PAPVD** of the right upper pulmonary vein (RUPV) may occur with the SVC type sinus venosus ASD. (A) Color Doppler shows RUPV (red) flow enter the confluence of LA, SVC, and RA, with ASD flow from the LA to RA (blue). (B) Post-repair, RUPV (arrow) drains into RA, while SVC flow from the RA.



# Ventricular Septal Defects

## Ventricular Septal Defects

**Types** (may be isolated or part of complex congenital heart disease)

- Perimembranous (80%): below AV and lateral to septal TV leaflet, small
- Muscular: any location in muscular portion of IVS, surrounded by myocardium multiple, small, and difficult to detect by 2D alone
- Inlet (AV canal): posterior to membranous IVS, between TV/MV, associated primum ASD, atrioventricular valve abnormality or complete AV canal defect
- Outlet (5–8%)(supracristal, subarterial, infundibular): RVOT portion above the crista terminalis anterior to membranous septum, below aortic and pulmonic valves

### 2D Imaging

- 2D views (see below), TTE better than TEE to image IVS
- Type, location, size
- Volume overload, dilates left-sided structures and PA:
  - LV size and function (see below)
  - LA dilated from ↑ return
  - Pulmonary artery dilatation and ± pulmonary hypertension
- RV less dilated, RVH if ↑ PAP or pressure overload in large VSDs
- IVS aneurysm may be detected, appears as “windsock”

### Doppler

- Color helps identify shunt location
- CW measure peak systolic pressure gradient between ventricles to classify as restricted/unrestricted (see below), direction of shunt (L→R or R→L)
- Estimate RVSP from VSD velocity and systolic BP (SBP), not TR jet  
 $RVSP = SBP - VSD \text{ gradient}$
- Shunt fraction Qp/Qs > 1.5, surgery recommended

VSD	Peak pressure (mmHg)	LA or LV dilatation	Pulmonary artery pressures
Restrictive	> 75	No	Normal
Mod restrictive	25–75	↑	↑
Nonrestrictive	< 25	↑↑	↑↑

VSD type	2D imaging/Best in	Doppler
Muscular	Difficult to 2D image, use color, multiple ME 4C, TG SAX	<b>Color</b> Flow disturbance on RV side with L to R shunts
Inlet (post to septal TV)	MV and TV in same plane ME 4C	
Perimembranous (A + S TV leaflets (R + non AV cusps)	LVOT below AV Extend to inlet, outlet, trabecular ME RVOT, 5C, AV LAX or SAX	<b>Spectral</b> CW shows high velocity L to R flow in systole
Outlet (below PV)	AV cusp herniation + AI ME RVOT, AV LAX	

What to tell the surgeon

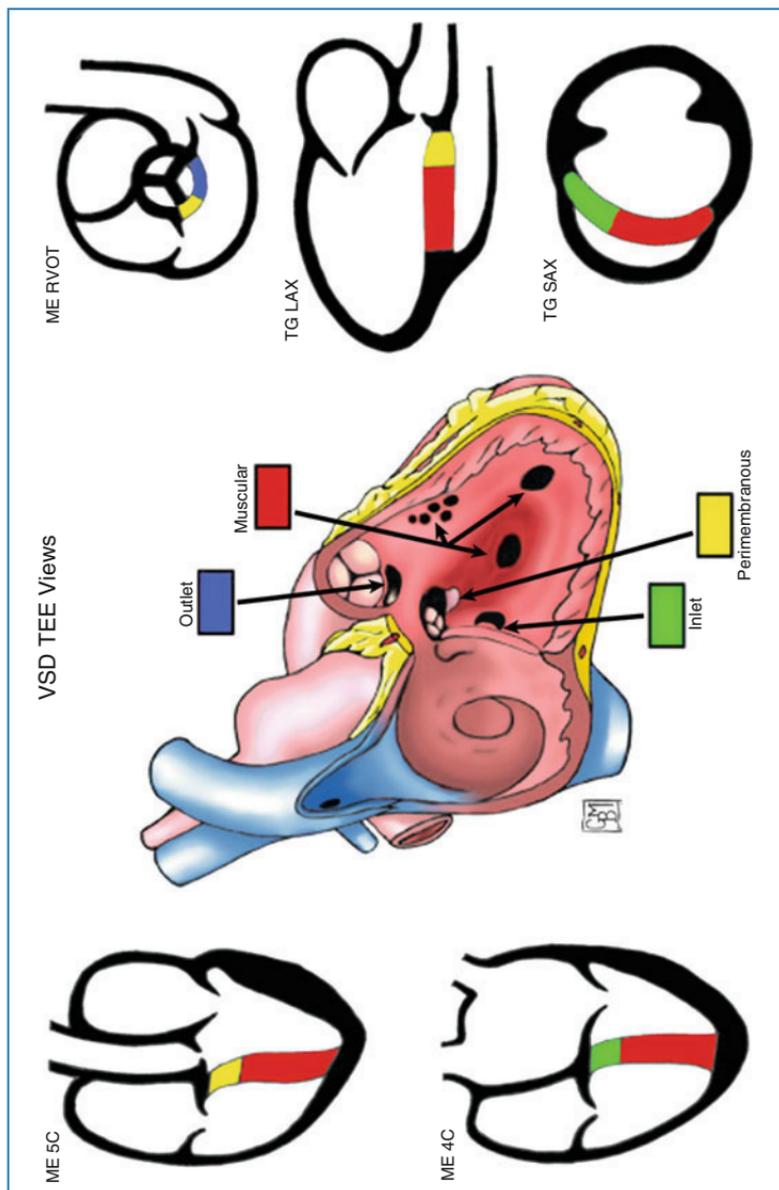
Pre-CPB:

- Location (type), size, number
- Shunt direction, peak pressure gradient
- Associated findings (RVH, RA, PASP)
- Associated pathology: complex congenital, AV cusp

Post-CPB:

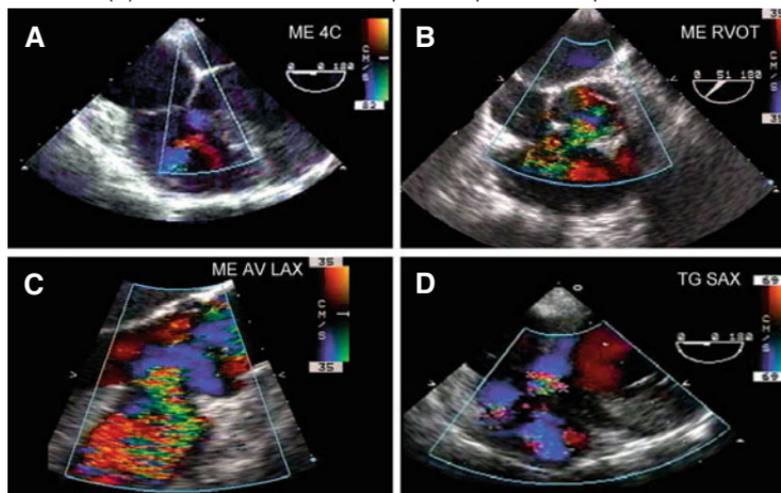
- Residual leak

## Ventricular Septal Defects

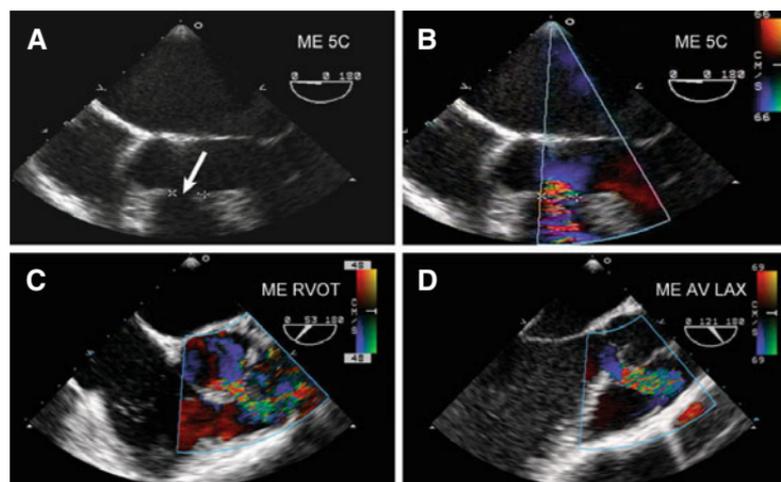


## Ventricular Septal Defects

**Perimembranous + Inlet VSD:** (A) ME 4C view with MV and TV at same level suggests an endocardial cushion defect. Color Doppler in ME (B) RVOT and (C) AV LAX views show mostly turbulent left to right flow through the VSD into the RVOT from the LV to RV. (D) TG SAX view shows flow in posterior part of the septum.

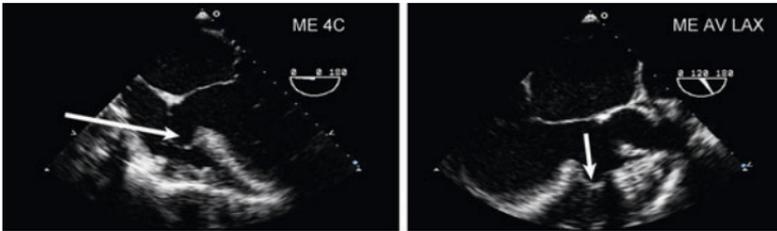


**Perimembranous + Outlet VSD:** (A) 2D ME 5C view (AV) shows a gap (arrow) in the IVS, which must be differentiated, from echo dropout. (B) Color Doppler shows mostly turbulent left to right flow through the VSD. (C) ME RVOT and (D) AV LAX views with color Doppler show flow is below the PV in the RVOT from LV to RV.



## Ventricular Septal Defects

**Aneurysm of membranous IVS** seen in ME 4C and AV LAX views.

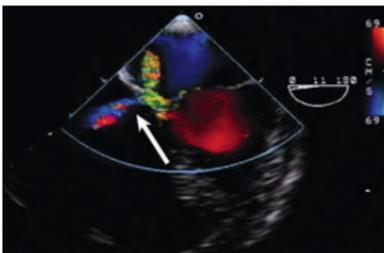
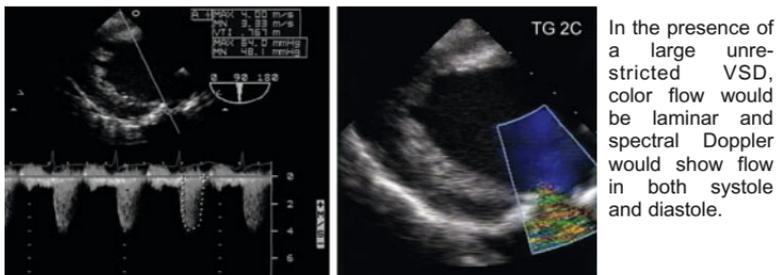


**Aortic valve cusp** prolapse through subarterial VSD with and without color Doppler.



### Doppler Flow through VSD

Perimembranous VSD imaged in TG 2C view allows spectral CW Doppler alignment. Turbulent flow suggests a restrictive VSD which using CW Doppler shows high velocity systolic flow from LV to RV, with a peak pressure gradient of 64 mmHg.



### Gerbode Defect

- Congenital: rare variant atrioventricular septal defect (AVSD)
- Acquired: post-MV surgery
- Shunt is directly between LV → RA
- Defect in superior portion of atrioventricular septum, between TV / MV
- Turbulent color flow with high CW Doppler pressure gradient
- Distinguish from perimembranous VSD flow (LV→RV) and TR

# Tetralogy of Fallot

## Tetralogy of Fallot

1. RV outflow tract obstruction (infundibular)
2. RV hypertrophy (RVH)
3. Over-riding aorta
4. Ventricular septal defect (VSD), large

## Associated Pathology

- ASD (Pentalogy of Fallot) (25%)
- Right-sided aortic arch (25%)
- Pulmonic valve atresia (10%)
- Second VSD (Down's syndrome)
- Coronary artery anomalies (10%)
- Systemic venous anomalies
- LVOT obstruction
- AV large (75%) with AI

## Previous Surgery

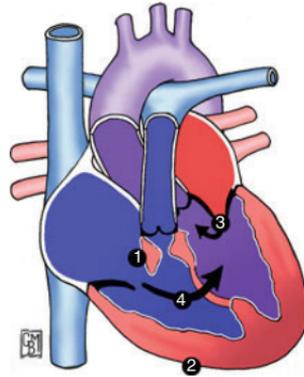
- Palliative shunt: Blalock-Taussig, Watterson, Pott's
- Close VSD
- Repair RVOT/PV: transannular patch, PV (valvotomy, replace)

## 2D Imaging

- Overriding aorta, VSD, RVH in ME AV LAX view
- VSD: large subaortic/membranous, unrestricted mixing
- Muscular RVOT dynamic obstruction, RVH, RV function
- Pulmonic valve stenosis (bicuspid, doming), annulus size, if dilated result in PI
- Check main PA size and branches, may be hypoplastic
- Large AV and root with AI
- May have ASD, anomalous coronaries (LAD arises from RCA crosses RVOT)

## Color/Spectral Doppler

- RVOT obstruction: ↑ velocity + turbulence at level of obstruction (valvular, subvalvular, supra-valvular)
  - Color or PW Doppler to locate the level of obstruction
  - CW to estimate peak pressure (> 80 mmHg)
- Pulmonic stenosis: peak + mean pressure gradients across pulmonic valve
- VSD pressure gradient (low as unrestricted), patch leak



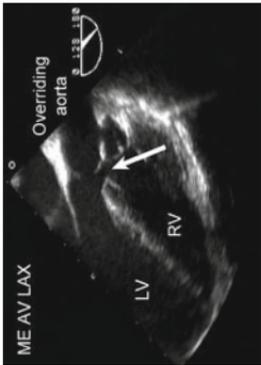
## What to tell the surgeon

- Uncorrected: VSD, RVOT level of obstruction, overriding aorta, RVH
- Corrected: VSD leak, pulmonic valve (PS/PI severity), RVOT obstruction
- RV size and function, aneurysmal RVOT if PI
- Associated findings: AI, LV function

## Post-CPB:

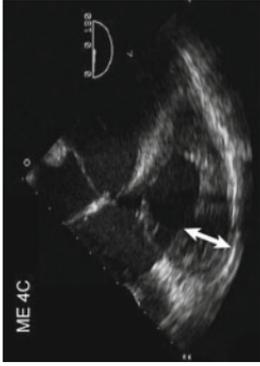
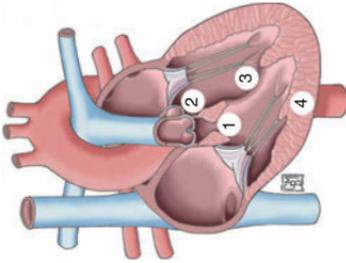
- Residual VSD patch leak
- Pulmonic valve function (prosthetic)
- RV size and contractility
- Residual RVOT obstruction
- TR severity

# Tetralogy of Fallot

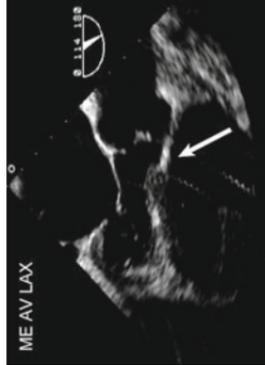


ME AV LAX view of an uncorrected TOF shows an unrestricted VSD (3, arrow) and aorta overriding the interventricular septum (2). Note the large AV. VSD patch closure is required.

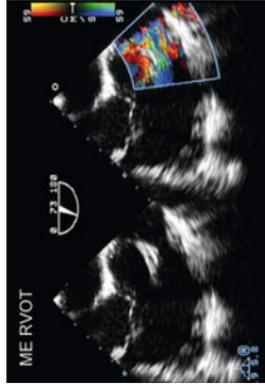
Tetralogy of Fallot



ME 4C view in TOF shows RV hypertrophy (4) from PS or RVOT obstruction. The free wall thickness is > 5 mm with a small cavity size. The overall RV systolic function is usually preserved.



The VSD patch (arrow) appears bright and echogenic without shadowing artifact. Examine using color Doppler for a residual patch leak.



The RVOT is narrowed due to trabecular hypertrophy (1) and antero-cephalad deviation of the outlet septum. Color Doppler shows turbulent RVOT flow.

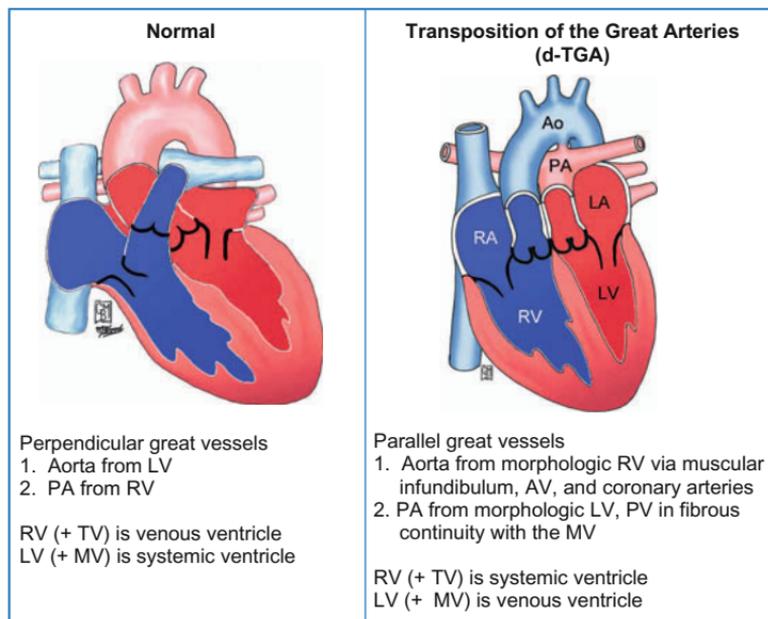


The PV may be stenosed (shown here) or insufficient. PV morphology is best seen in RVOT or UE arch SAX view, which also allows good spectral Doppler alignment.

## D–Transposition of the Great Arteries

### Transposition of the Great Arteries (D-TGA)

The pulmonary artery arises from LV and the anterior aorta (with coronary arteries) from RV. This arrangement forms two parallel circulations, requires an ASD or VSD for survival. The atria, atrioventricular valves, and ventricles are all positioned normally.



**Associated pathology:** ASD, VSD, obstructed pulmonary outflow, AV valve abnormalities, coronary artery, and aortic arch anomalies.

**Surgical interventions:** Mustard or Senning (atrial baffles), Jatene (arterial switch)

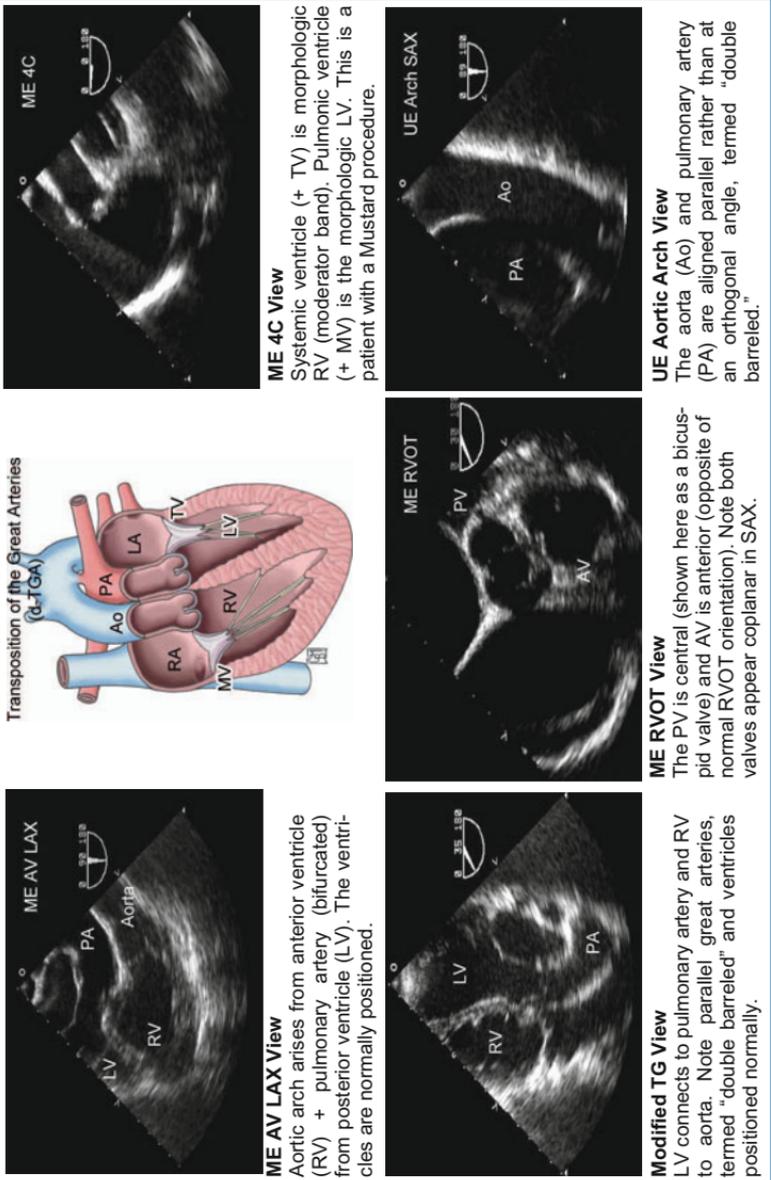
### 2D Imaging

- Parallel great vessels, “double barreled”
- Systemic (morphologic RV) ventricle size and function: dilated, RVH
- Venous (morphologic LV) ventricle: smaller, banana shaped, IVS bulges into LV
- Systemic atrioventricular valve (TV) regurgitation
- Assess PAP from venous atrioventricular valve (MR jet)
- Exclude: LVOT obstruction from bowing of IVS into LVOT and low resistance PA SAM  
Premature closure pulmonic (systemic) valve  
LVOT turbulence

### Color/Spectral Doppler

- Atrioventricular valve leaks (MR, TR)
- Baffle leaks (see pg. 168)
- Baffle obstruction

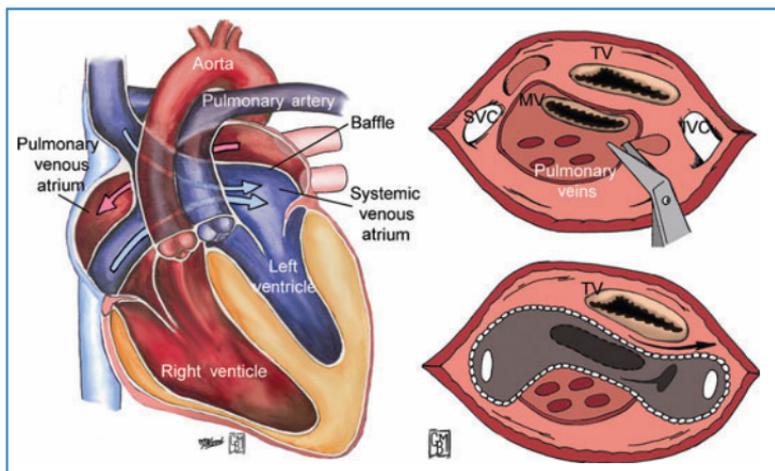
## D-Transposition of the Great Arteries



## Mustard Procedure

### Mustard Procedure

Atrial switch (Mustard or Senning) procedure replaces the inter-atrial septum (IAS) with baffles that redirect blood flow to the ventricles.



The IAS is excised; the coronary sinus drains into the LA. A pericardial patch is sutured to allow drainage of the pulmonary veins into the pulmonary venous atrium and outflow through the tricuspid valve (TV) into the RV. The SVC, IVC, and CS drain into the systemic venous atrium with outflow through the mitral valve (MV) into the LV.

### Baffle Obstructions

#### Systemic Baffle (Venous or Caval)

- Usually occurs at the junction SVC and RA
- SVC is dilated
- Color Doppler continuous turbulent flow, loss of respiratory variation
- PW continuous (nonphasic) flow, suspicious if  $>1.2$  m/s, more convincing  $>1.5$  m/s
- Contrast injection in upper extremity, image the IVC
  - No obstruction: contrast fills the systemic venous atrium (SVA) from above and the IVC remains free of contrast
  - Partial obstruction: normal filling of SVC with gradual appearance of contrast material in the IVC from collaterals
  - Complete obstruction: SVC fills with contrast only from below by collaterals

#### Pulmonary Venous Baffle

- Usually mid-baffle or isolated pulmonary vein stenosis
- Color Doppler lower velocity turbulent flow does not rule it out
- PW/CW: Diastolic velocity convincing if  $> 1.5$  m/s, loss of phasic flow (pattern as normal pulmonary vein flow)

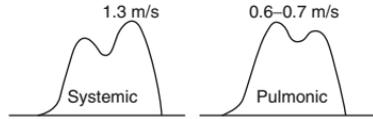
## Mustard Procedure

Baffles are not imaged at the same level; advance and withdraw the TEE probe.

Baffle flows normally show:

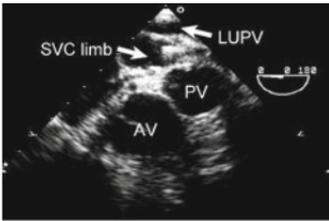
- Velocity (low)
- Phasic flow
- Respiratory variations

- Baffle obstruction have high velocity, nonphasic flow without respiratory variation.
- Baffles leaks are very difficult to diagnose. May see color across baffle walls and can be confirmed with contrast as previously described.



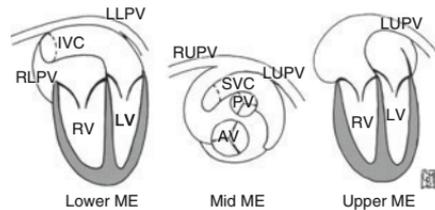
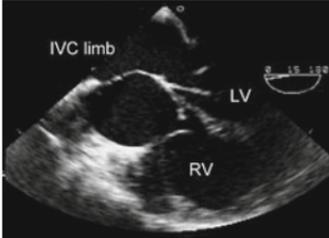
### Systemic Venous Baffle

This baffle returns systemic venous blood from the SVC and IVC to the subpulmonic ventricle (morphologic LV) which supplies blood via the PA to the lungs.



The SVC forms the upper limb of the systemic venous baffle and is imaged in a mid ME view. It appears in the middle of the display and may have pacer wires or catheters making it easier to identify.

The IVC forms the lower limb of the systemic venous baffle and is imaged at gastro-esophageal junction (lower ME) near the liver.

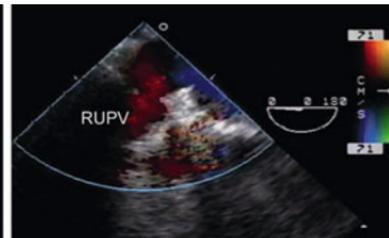
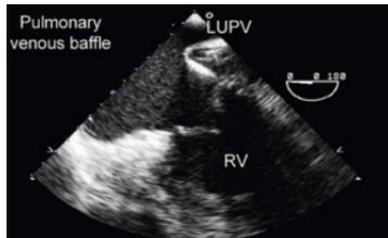


### Pulmonary Venous Baffle

This baffle returns oxygenated blood from the pulmonary veins to the systemic ventricle (morphologic RV) which supplies the aorta.

Left pulmonary veins: LUPV imaged in usual position at 0°–60°, above (posterior) to the SVC baffle (upper ME).

Right pulmonary veins: RUPV and RLPV are imaged at 0°–30° in their usual position.



# Fontan Procedure

## Fontan

Fontan circulation allows venous blood to enter the PA usually without a subpulmonic ventricle. Palliative procedure for an univentricular circulation.

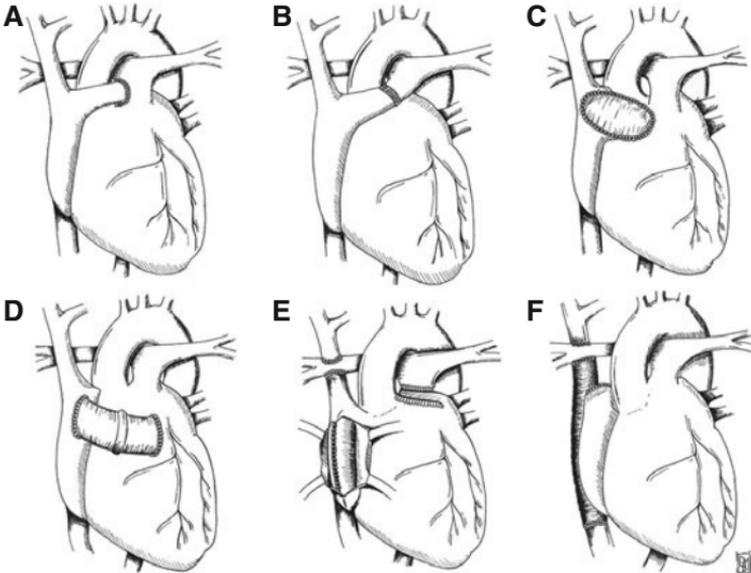
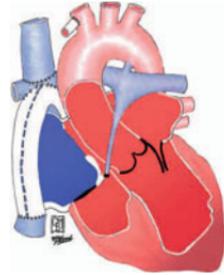
There are many variants:

### Classic Fontan

- (A) RA to PA directly, side of main PA
- (B) RA to PA directly, end of main PA

### Modified Fontan

- (C) RA to RV via pericardial patch
- (D) RA to RV via conduit ( $\pm$  valve)
- (E) Total cavopulmonary anastomosis (IVC+SVC+RA) with intra-atrial (lateral tunnel)
- (F) Total cavopulmonary anastomosis (IVC+SVC+RA) with extra-cardiac conduit

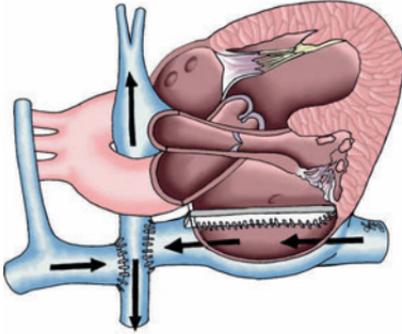


### TEE Imaging

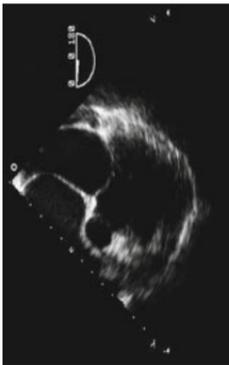
- Hypoplastic RV, assess RV size and function
- Residual atrial leak
- Assess ventricular end diastolic diameter and contraction
- Systemic atrioventricular valve regurgitation that may reflect ventricular dilatation and dysfunction
- Pulmonary artery and vein Doppler
- Fontan circulation: Doppler profile, velocity, respiratory variation, mass/thrombus

# Fontan Procedure

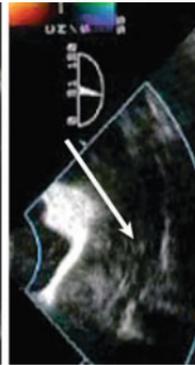
Classic Fontan + Bidirectional Glenn



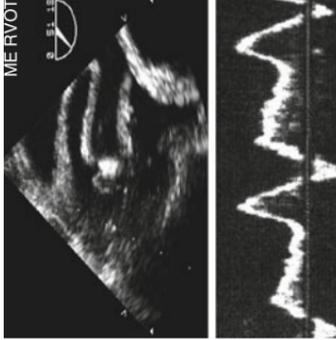
Patient with a single ventricle and two atria imaged in ME 4C view.



Thrombosed Fontan (arrow) in LAX, SAX.



Tricuspid Atresia in ME 4C view at right.

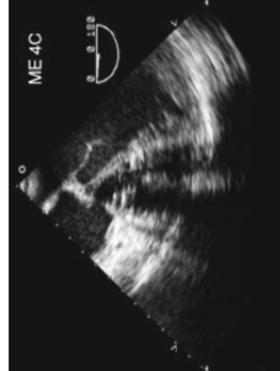


## PW Doppler Trace (PA conduit in RA)

- Low velocity  $\leq 1$  m/s
- Bi (or tri) phasic flow
  - Forward flow in atrial systole
  - Retrograde flow in early atrial relaxation followed by
  - Lower velocity forward flow
- Respiratory variation,  $\uparrow$  inspiration

## Obstruction of atrio-pulmonary if:

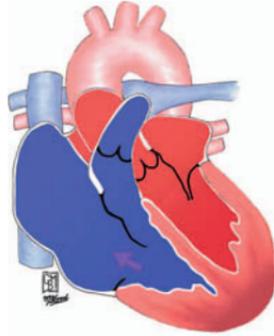
- High velocity  $> 1$  m/s
- Turbulent
- Continuous flow
- Loss of respiratory variation



# Ebstein's Anomaly

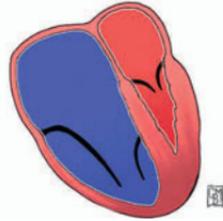
## Ebstein's Anomaly

- Congenital abnormality of the tricuspid valve
- Progressive displacement of septal and posterior leaflets toward the RV apex
- Leaflets originate from RV wall, "tethered" to RV
- Dysplastic TV leaflets: large malformed anterior leaflet, hypoplastic septal leaflet
- TR originates below the annulus in the RV, variable severity
- Atrialization of RV (dilated), small functional RV
- RA enlarged
- Associated: ASD, L-TGA, mitral valve prolapse, PFO, WPW
- Surgical repair includes: mobilize anterior TV leaflet + plicate RV + TV annuloplasty



## TEE Imaging (ME 4C view)

- Dilated RA
- Dilated TV annulus
- Enlarged atrialized RV + small functional RV
- Large malformed anterior leaflet tethered to RV wall
- Apically displaced hypoplastic septal leaflet with increased distance from the MV annulus > 20 mm or  $\geq 8 \text{ mm/m}^2$
- Severe TR, origin below the TV annulus



### 2D echo features

Tethered ant leaflet (severe)	3
Tethered ant leaflet (mild)	1
Restricted motion ant leaflet	2
Functional RV < 35%	2
Absent septal leaflet	1
Displaced ant leaflet/Aneurysmal RVOT	1
RA diameter > 60 mm/mm <sup>2</sup>	1
Severe tricuspid prolapse	1

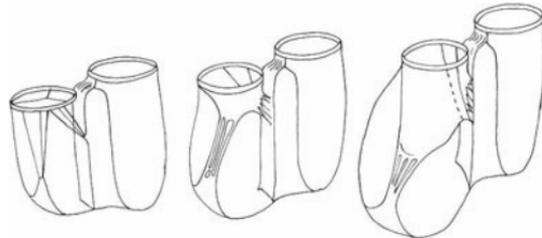
Index > 5 predicts need for TV replacement as compared to repair.

### Surgical repair principles

- Large untethered anterior leaflet mobilized to small septal leaflet
- Reduce TR
- Plicate atrialized RV
- Reduce RA size

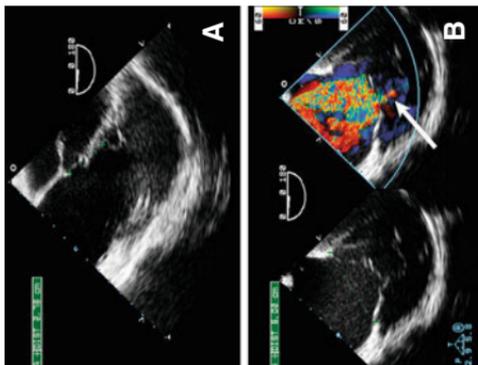
Source: Shiina A, et al. *Circulation* 1983; 68:534-44.

Progression of TV septal leaflet displacement and elongation of the anterior leaflet. The septal leaflet is tethered to the RV walls.

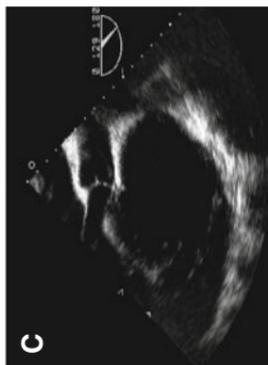
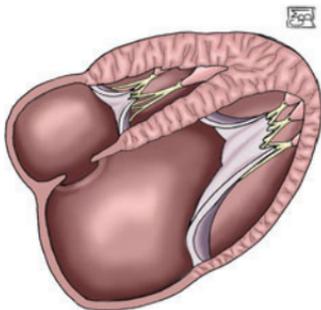


## Ebstein's Anomaly

(A) ME 4C view shows an apically displaced TV septal leaflet, as measured from the MV annulus in diastole. The functional RV is small; the remainder of the RV is atrialized. (B) The TV annulus measured during systole is severely dilated. The severe tricuspid regurgitant jet (arrow) starts well below the TV annulus at the leaflet coaptation point.



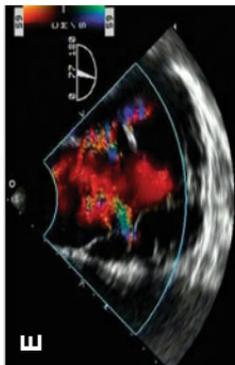
## Ebstein's Anomaly



(C) ME LAX view demonstrates a large atrialized RV in diastole. This requires an image depth of 18 cm to show the entire RV.

(D) The ME RVOT view shows tethering of tricuspid valve leaflets.

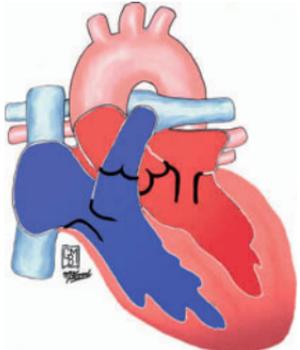
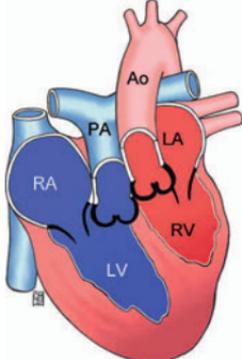
(E) Laminar retrograde flow across the tricuspid valve is suggestive of severe TR with significant RV dysfunction.



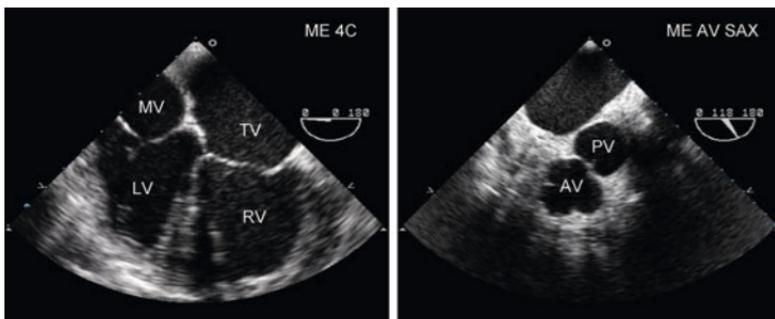
## L-Transposition of the Great Arteries

### Congenitally Corrected Transposition of the Great Arteries (L-TGA)

The morphologic LV is the venous ventricle and gives rise to the PA. The morphologic RV is the systemic ventricle and gives rise to the aorta. Have atrioventricular discordance + ventriculo-arterial discordance so "2 wrongs make a right". The morphologic RV was not intended to support the systemic circulation. Patients are usually asymptomatic until they have systemic ventricular (RV) failure and atrioventricular valve (TV) regurgitation.

Normal	L-Transposition of the Great Arteries
	
<ol style="list-style-type: none"> <li>1. Perpendicular great vessels</li> <li>2. PA is anterior to aorta</li> <li>3. RV (+ TV) is venous ventricle, PA from RV</li> <li>4. LV (+ MV) is systemic ventricle, aorta from LV</li> </ol>	<ol style="list-style-type: none"> <li>1. Parallel great vessels</li> <li>2. Aorta is anterior to PA</li> <li>3. LV (+ MV) is venous ventricle, PA from LV</li> <li>4. RV (+ TV) is systemic ventricle, aorta from RV</li> </ol>

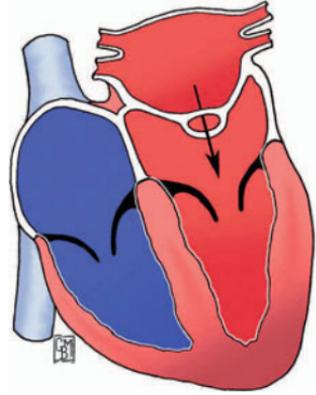
The ME 4C view shows the morphologic RV on the right side of the display. The TV is apically displaced and is frequently regurgitant. The ME AV SAX view shows the aortic and pulmonic valves are coplanar, rather than being orthogonal.



## Cor Triatriatum

### Cor Triatriatum

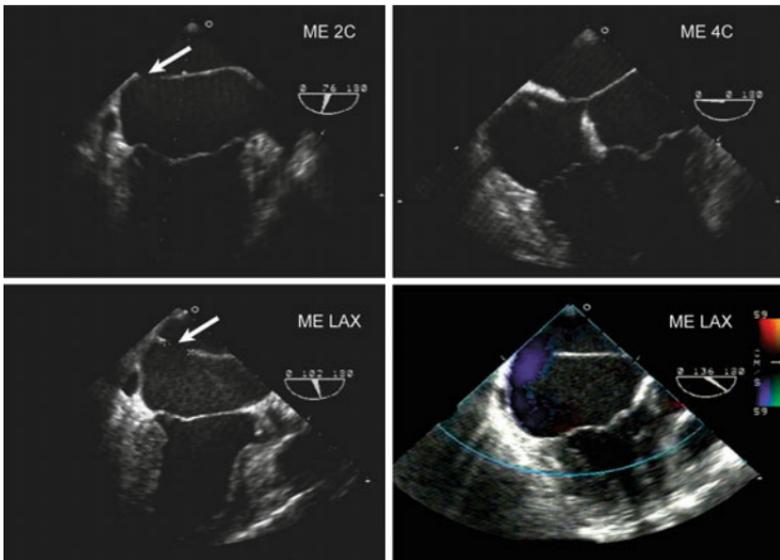
- Intra-atrial membrane divides LA into two parts:
  1. Accessory pulmonary venous chamber into which the pulmonary veins drain
  2. LA chamber contiguous with MV
- The connection between the accessory chamber and true LA varies in size and may produce pulmonary vein obstruction.



### TEE Imaging

- Intra-atrial membrane seen in multiple views, inserts proximal to LAA into the "coumadin ridge"
- Diastolic movement towards the MV
- May have RVH + RV dilatation
- May have associated PFO/ASD, persistent left SVC, AVSD, PAPVD, coarctation
- Color flow laminar or turbulent flow
- PW Doppler pressure gradient, significant if mean gradient > 10–12 mmHg

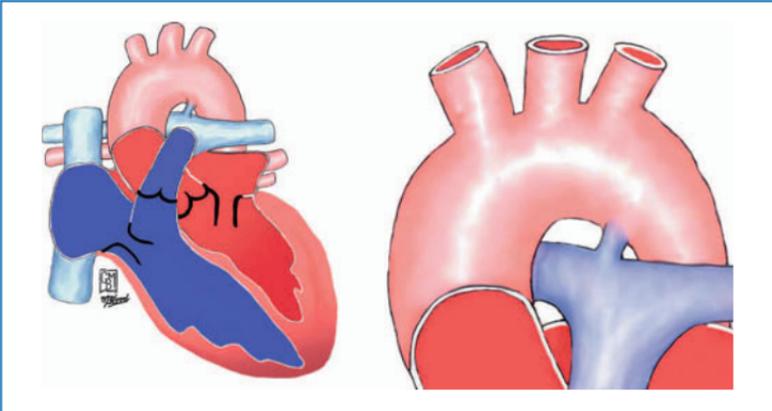
Intra-atrial membrane seen in the LA in multiple views, ME 2C view above the LAA, ME 4C view attached to the IAS. The ME LAX and ME 2C views show a gap in the intra-atrial membrane with laminar color flow. The membrane, an incidental finding, was resected at the time of surgery.



# Patent Ductus Arteriosus

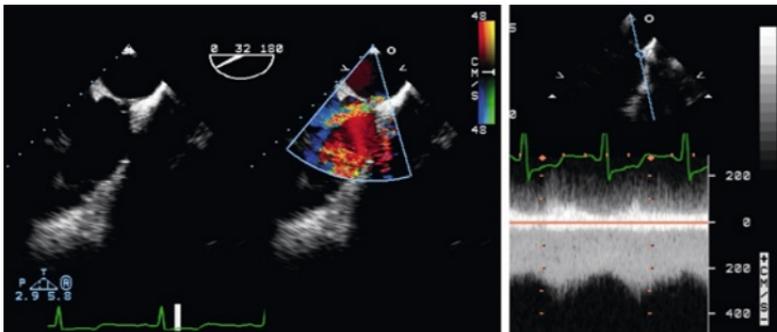
## Patent Ductus Arteriosus

PDA is persistence beyond 10 days after birth of the normal fetal connection between the pulmonary artery (PA) and descending aorta. In utero it enables blood to bypass the lungs and perfuse the fetus. It usually closes spontaneously at birth. PDA is an uncommon isolated pathology but may be present in complex congenital heart disease. If untreated, there is an increased risk of endocarditis and overall mortality. Treatment includes open surgical closure and percutaneous device closure.



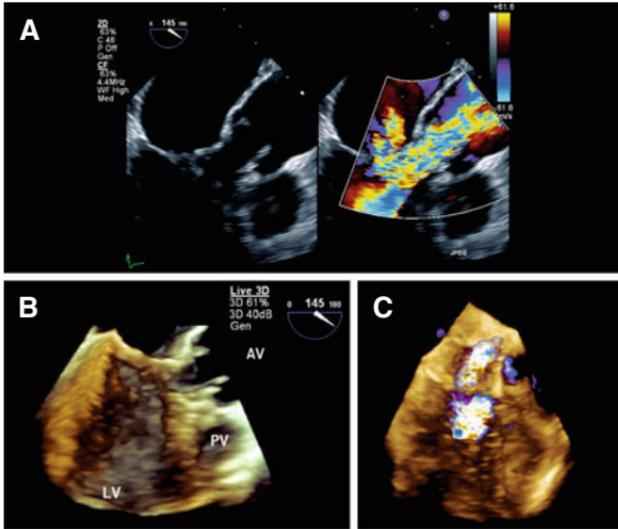
1. Connection between PA and descending aorta at the level of the left subclavian artery. Variable in size though usually restrictive with mosaic color.
2. RV often dilated.
3. PA may be dilated.
4. LV only dilated if large PDA.
5. RVSP estimates pulmonary hypertension.

UE Aortic Arch SAX color compare view shows turbulent flow from the aorta to the main pulmonary artery. The connection between both structures is seen in the 2D image. CW Doppler shows continuous high velocity systolic and diastolic flow. Bidirectional flow suggests elevated PA pressures compatible with Eisenmenger's.

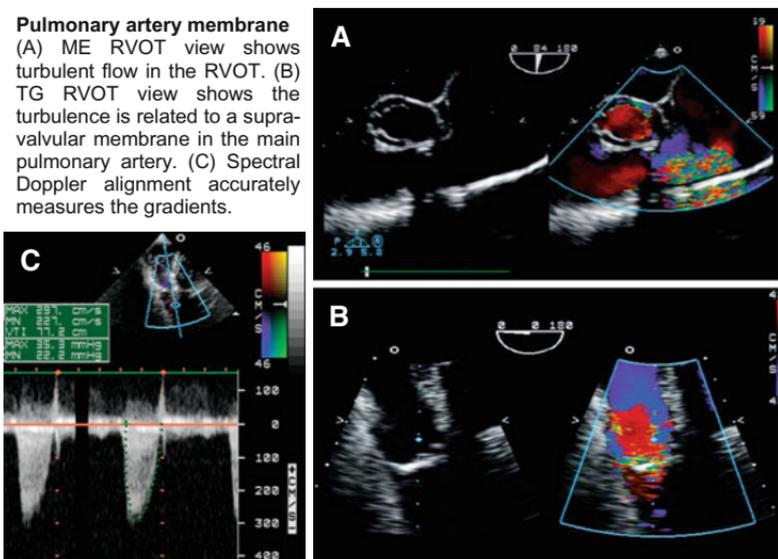


## Subaortic Membrane

**Subaortic membrane** (A) ME AV LAX views show turbulent flow starting below the AV in the LVOT. (B) The narrowed LVOT is seen en-face in a 3D Live view of the LVOT and (C) the turbulent flow (with MR) is shown with 3D color full volume view.



**Pulmonary artery membrane** (A) ME RVOT view shows turbulent flow in the RVOT. (B) TG RVOT view shows the turbulence is related to a supra-valvular membrane in the main pulmonary artery. (C) Spectral Doppler alignment accurately measures the gradients.



# Variants, Foreign Material, Masses and Endocarditis

Normal Variants.....	180–182
Foreign Material.....	183–184
Masses Tumor.....	185–189
Masses Thrombi.....	190–191
Endocarditis Vegetations .....	192–193
Endocarditis Complications.....	194–197

# Normal Variants

## Normal Variants

### Left Atrium

- Coumadin ridge (LAA/LUPV): echogenic "Q" tip shape
- Pectinate muscles: trabeculations in LAA
- Dilated coronary sinus (> 1 cm), seen posterior in LA groove
  - Ddx: persistent LSVC, high right-sided pressures
- Persistent left SVC (see pg. 182)
  - Drains into dilated coronary sinus (> 2 cm)
  - Saline contrast in left arm shows bubbles in LSVC + coronary sinus before RA

### Right Atrium

- Crista terminalis (SVC/RA): muscle ridge
- Eustachian valve (IVC/RA): valve channels blood from RA through PFO
- Chiari network: remnant of sinus venosus, arises from Eustachian valve
  - Fine filaments, mobile
  - Associated with PFO, IAS aneurysm, paradoxical emboli
- Pectinate muscles are not isolated to RAA but extend to the vestibule
- Thebesius valve: valve to coronary sinus, prevents regurgitation of blood into the coronary sinus

### Left Ventricle

- Papillary muscles: normally two, abnormal if one (parachute MV)
- Aberrant chordae
- False tendons: fine filaments that may represent false chordae

### Right Ventricle

- Moderator band: prominent apical muscle band from septum to anterior PM
- Trabeculations: muscle bands in the RV (prominent in RV hypertrophy)
- Papillary muscles usually three are present

### Aortic Valve

- Nodules of Arantius: points of coaptation AV cusps
- Lambl's excrescences: degenerative strands on either side of valve cusps

### Mitral Valve

- Redundant chordae

### Pericardium

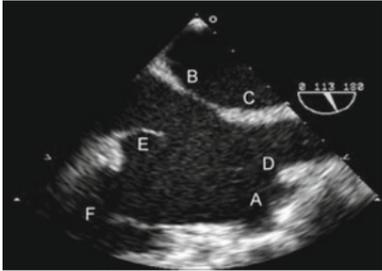
- Adipose tissue: epicardial fat pad
- Transverse sinus: space between posterior wall of ascending aorta and anterior LA (RVOT view). May appear as cystic mass differentiate from LAA, fibrin, or cyst.

### Inter-atrial Septum (see pg. 155)

- Lipomatous hypertrophy IAS: echogenic "dumb bell shaped" IAS
- IAS aneurysm: mobile septum > 1.0 x 1.0 cm into atria, sigmoid shape
  - ↑ risk of stroke due to thrombus formation
  - 50% PFO

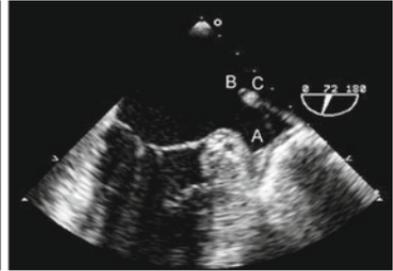
## Normal Variants

Artifacts are any structure in an image that does not match an anatomical tissue structure. Pitfalls are errors in interpretation of an artifact or normal structures that mimic pathology and may provoke unnecessary clinical interventions. It may be difficult to differentiate normal variants from common pathological findings and artifacts. Knowledge of normal variants and careful imaging in multiple planes may help.



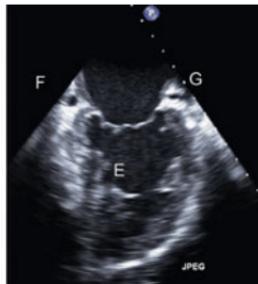
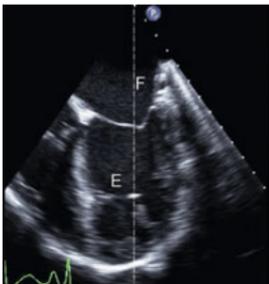
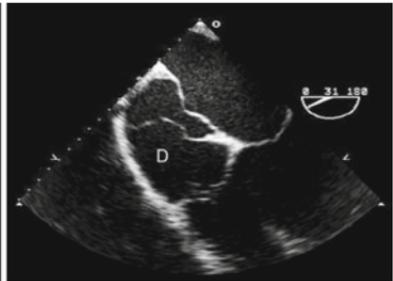
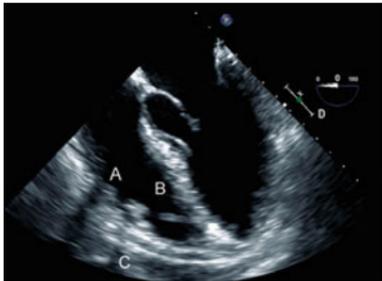
### Modified Bicaval View (110°)

- A. Pectinate muscle RAA
- B. IAS foramen ovalis
- C. Lipomatous hypertrophy IAS
- D. Crista terminalis (SVC/RA)
- E. Eustachian valve or Chiari network (IVC/RA)
- F. Tricuspid valve



### LAA View (70°)

- A. LAA pectinate muscle
- B. Coumadin ridge
- C. Left upper pulmonary vein (LUPV)
- D. Persistent LSVC (see pg. 168)



### ME 4C View (0°–30°)

- A. RV trabeculations
- B. Moderator band
- C. Epicardial fat
- D. Chiari network
- E. LV false tendons

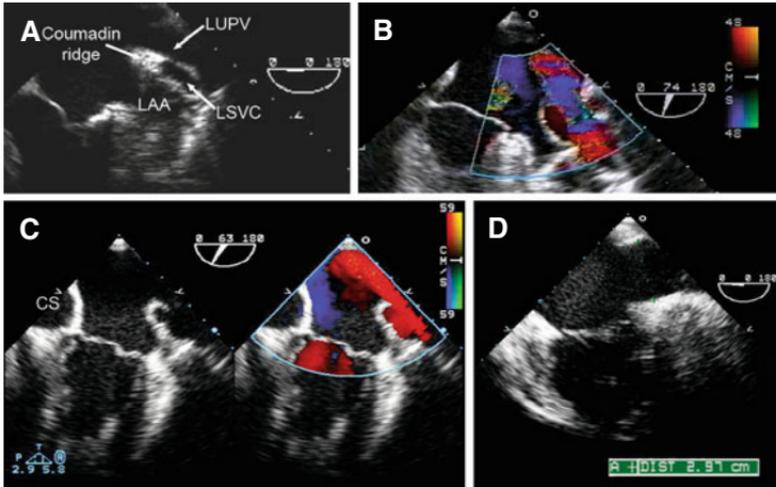
### ME 2C (90°)

- F. Coronary sinus
- G. Circumflex artery

## Normal Variants

### Persistent Left Superior Vena Cava (LSVC)

A persistent LSVC results from failure of the left posterior cardinal vein to reabsorb and thus connects directly to the coronary sinus (CS). (A) This is imaged as a cystic structure between the LUPV and left atrial appendage (LAA) in the ME LAA view. Note there appears to be three cystic structures: the LUPV, persistent LSVC, and LAA. (B,C) Color Doppler identifies flow in the structure. (D) The CS is dilated (> 2 cm) as seen in (D) LAX in the lower esophageal Coronary Sinus view or in (C) SAX in the ME 2C view. Agitated saline injected into the left arm enters from the left subclavian vein and rapidly appears in the CS. A Swan-Ganz catheter inserted in the left veins may also appear in the CS.

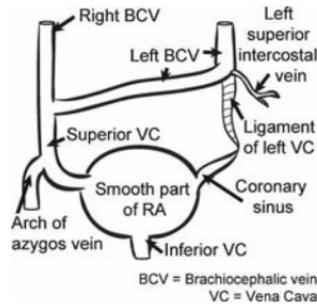


### Different subtypes of persistent LSVC exist:

- Majority have both R and L SVC present.
- Rarely there is absence of the R SVC.
- A bridging innominate vein may be absent in 65% of patients.
- In 80–90% of patients, the persistent LSVC drains into the CS which enters the RA. It may however drain into the LA resulting in a L→R shunt.

### Differential of dilated coronary sinus:

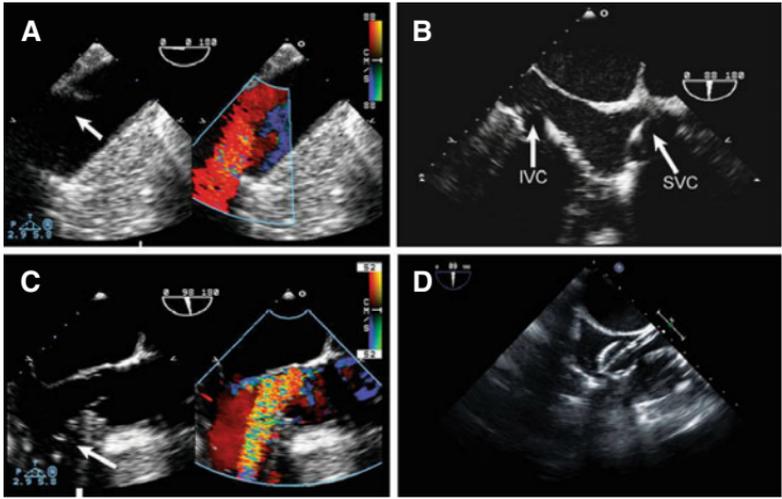
- Persistent LSVC
- Elevated RAP
- Coronary arterio-venous fistula
- Partial anomalous pulmonary venous drainage
- Unroofed coronary sinus (LA→CS flow)



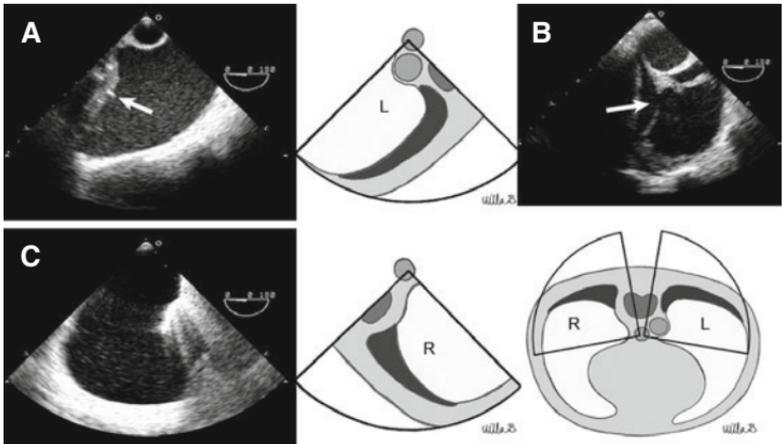
Source: Goyal SK, et al. *Cardiovasc Ultrasound* 2008; 6:50.

## Foreign Material

**Cannulation:** (A) Aortic cannula (arrow) and flow in distal ascending aorta. (B) SVC and IVC cannula in situ for bicaval cannulation seen in bicaval view. (C) Aortic cannula placed in the LV apex in a patient with an aortic dissection (arrow). (D) Coronary sinus cannula is seen in the SVC/right atrium and is directed to the coronary sinus.



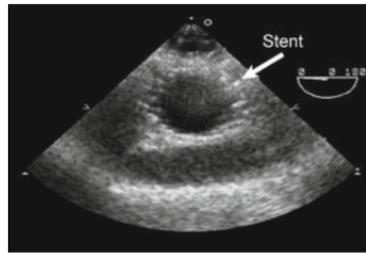
**Pleural Effusions:** (A) Left pleural effusion appears as an echolucent region immediately below the descending aorta. The “tiger’s claw” is directed left. Note left lung atelectasis (arrow). (B) Combined small pericardial effusion (arrow) medial to the L pleural effusion immediately below the aorta. (C) Right pleural effusion is an echolucent space with the “tiger’s claw” directed right above the liver. No aorta is present.



## Foreign Material

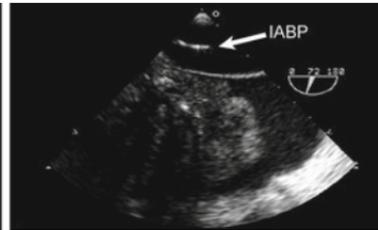
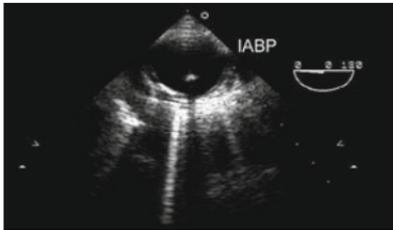
### Coarctation Stent

Look in proximal descending aorta and arch. Appears as a circular cluster of echogenic dots comprising the edges of the stent.



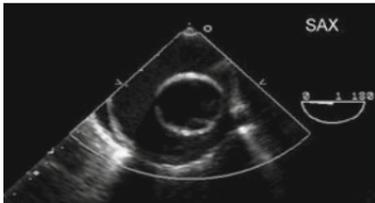
### Intra-aortic Balloon Pump Catheter

The IAB catheter tip is seen in Descending Aortic SAX and LAX views. Optimal IAB position is just below the left subclavian artery in the descending thoracic aorta imaged in both SAX and LAX.



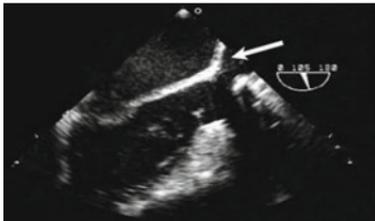
### Elephant Trunk

The first stage of the procedure in a patient with mega-aorta syndrome involves replacement of the AV, ascending aorta and arch with a prosthetic valve and Dacron graft. Dacron graft is imaged within the descending aorta in SAX and LAX views and appears free floating in the dilated native aorta. The proximal end is attached to the distal aortic arch, and the distal end is left unattached. The second stage uses an endovascular or open approach to secure the distal Dacron graft.



### Pulmonary Artery Band

Band placed on main PA in patient with L-TGA. Note distal turbulent flow after band.



## Masses Tumor

### Masses

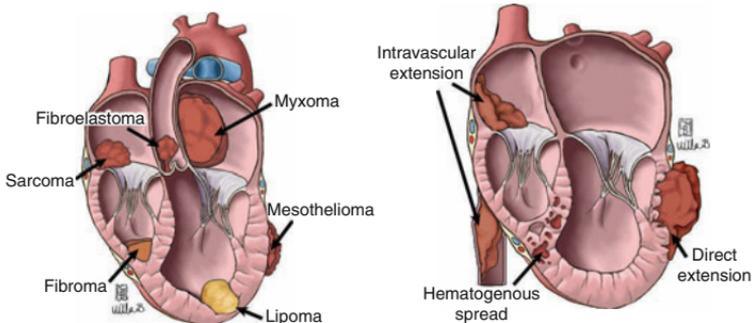
- Normal variants (see pgs. 180–182)
- Masses are abnormal structures within or adjacent to the heart
- Etiology includes:
  - Thrombus: located on pacer wires and catheters or in LAA, LV, as discrete spherical or laminar lesions often associated with LV dysfunction or atrial fibrillation
  - Vegetations: located on valves, myocardium, foreign material. Typically echogenic irregular independently mobile with associated valvular regurgitation
  - Cardiac Tumors (Source: Tazelaar HD, et al. Mayo Clin Proceed 1992;67:957-65.)
    - Primary cardiac tumors are rare 0.03%, most are metastatic 1%
    - 1° benign tumors (75%)
      - Myxoma (30%): LA > RA > RV = LV
      - Lipoma (10%): LV, RA, IAS
      - Papillary fibroelastoma (9%): AV > MV > TV
      - Fibroma (4%): LV > RV, IVS
    - 1° malignant tumors (25%)
      - Angiosarcoma (9%): RA, pericardium
      - Rhabdomyosarcoma (6%)
      - Mesothelioma (2%)
      - Fibrosarcoma (1%)
    - 2° metastatic:
      - Direct extension: lung, esophagus, breast
      - Intravascular: SVC (bronchogenic, thyroid), IVC (renal, hepatoma)
      - Hematogenous: lymphoma, melanoma, leukemia

### Diagnosis

- Echocardiography, CT, MRI
- Location (single or multiple, site of attachment or direct extension)
- Size
- Mobility
- Effect: obstruction, LV dysfunction, atrial fibrillation, emboli

### Surgery

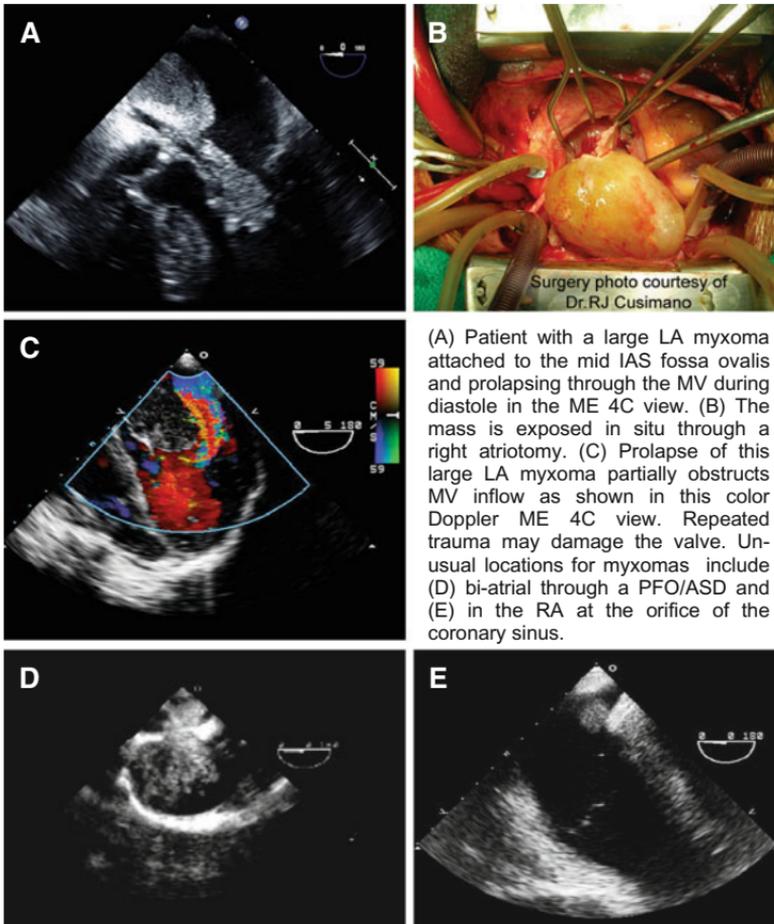
- For diagnosis or excision
- Require complete excision and reconstruction if needed
- Avoid tumor manipulation to prevent embolization



## Masses Tumor

**Myxomas** are the commonest primary cardiac tumor.

- Gelatinous mucoid texture composed of mural endocardial cells in myxomatous stroma. Typically appear as irregular polypoid, pedunculated, or short broad-based attachment and are of variable size.
- Location: LA > RA > RV = LV, majority solitary, multiple (3–5%)
  - Vacuulations may appear as echolucent areas from tumor necrosis
  - Independently mobile, deforms during the cardiac cycle
- Result in valve dysfunction (obstruction, regurgitation) or embolization (LA 30–40%, LV 50%)
- Syndrome myxoma = Carney's complex: familial, multiple sites



## Masses Tumor

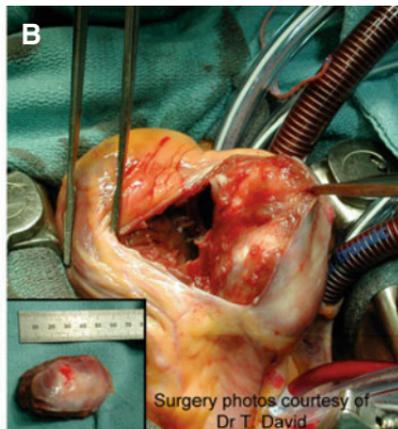
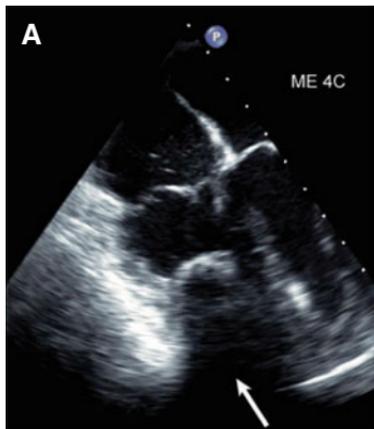
**Fibroelastomas** are the second commonest cardiac tumor and the commonest valve tumor. Gross pathology resembles sea anemone with frond-like projections. Involve semilunar valves (ventricular side): AV (44%), PV (8%) and atrioventricular valves (atrial side): MV (35%), TV (15%). Commonly presents as embolus. Surgery almost always indicated, involves local excision with underlying margin and valve repair.



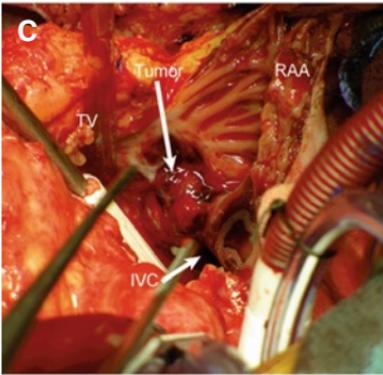
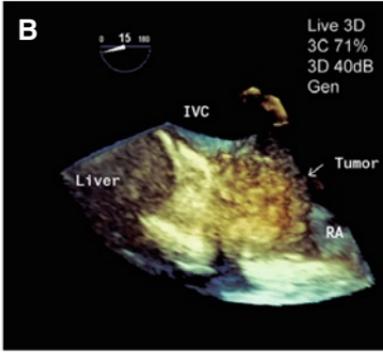
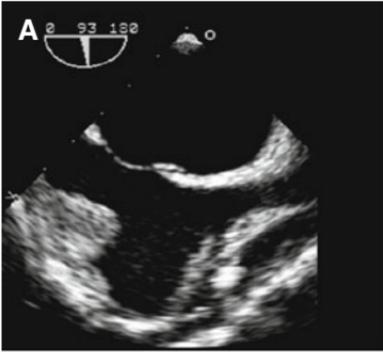
- Pedunculated
  - Small size (1 cm)
  - Pom-pom appearance
  - Narrow stalk
  - Mobile
- Homogeneous speckled appearance
- Stippled edge from fronds
  - Mobile undulating edges



This patient presented with a RV tumor and underwent complete resection requiring RV reconstruction with a pericardial patch. Tumor extension into the RV as a hypo-echoic mass is seen in (A) ME 4C view and (B) at the time of surgery. The anterior papillary muscle had to be resected and reimplanted. Pathology confirmed a fibroma.

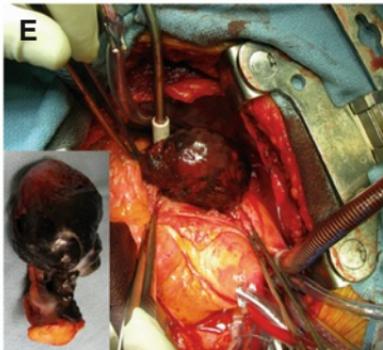
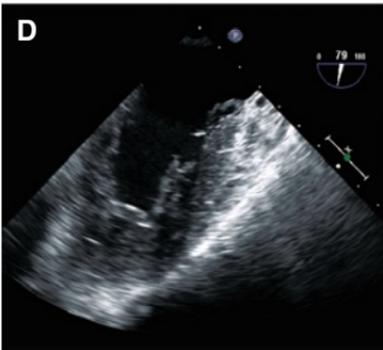


**Masses Tumor**



Patient with a RA sarcoma at the junction of the inferior vena cava (IVC). The tumor location in the RA relative to the IVC is best shown in the (A) 2D ME bicaval and (B) Live 3D bicaval views. (C) The tumor is seen in situ at the time of surgery through a right atriotomy. Resection of the tumor and part of the IVC and RA was performed under circulatory arrest.

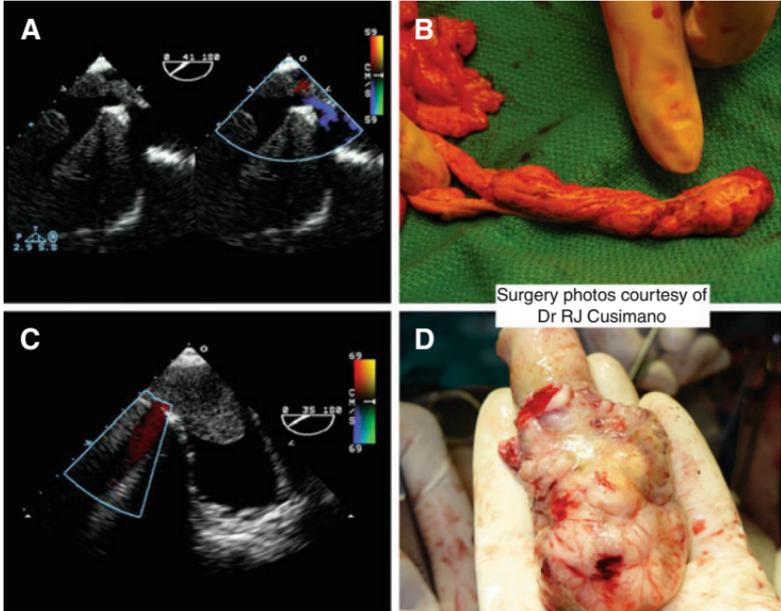
Surgical photos courtesy of Dr. RJ Cusimano.



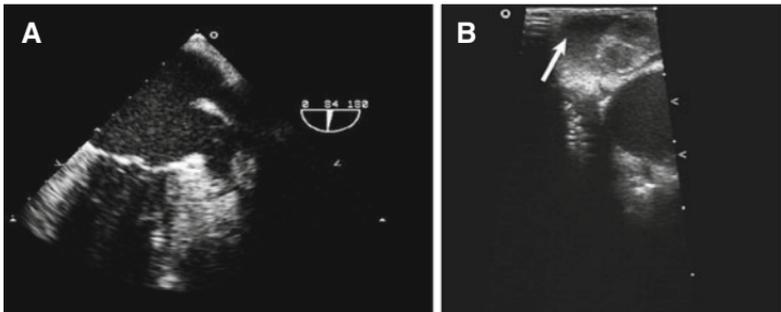
(D) Patient with metastatic melanoma of the LV apex. The ME 2C view shows a fullness in the LV apex with similar consistency to surrounding myocardium. (E) At the time of surgery the tumor was remarkably well encapsulated.

## Masses Tumor

Patient with a renal cell tumor extending via the IVC to the RA junction. (A) Color Doppler does not show obstruction in the IVC at the RA junction. (B) The right kidney with tumor extension was removed without requiring CPB. (C) A large leiomyosarcoma of the IVC extends into the RA as shown in this color Doppler hepatic vein view. (D) Surgery required extensive resection and reconstruction of IVC.



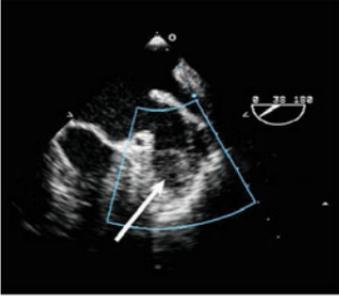
Patient with sarcoma of the upper lobe of the left lung. MRI suggested extension into the LUPV. (A) Imaging of the LUPV in the ME 2C view did not show any tumor in the proximal LUPV. (B) Epicardial exam directly on the more distal portion of the LUPV demonstrates the tumor (arrow).



## Masses Thrombi

Cardiac thrombi result from either primary cardiac, hematologic, or rheumatologic (Behcet's syndrome) etiologies. Thrombi can form:

- In an area of stasis: LA (MS, atrial fibrillation), LV (abnormal wall motion)
- On a catheter or device related (RA, RV)
- As thrombus in transit (RA, RV, PA)

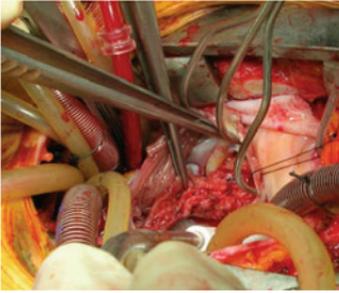


### LAA Thrombus

- LA enlarged + spontaneous echo contrast
  - Highest incidence in MS and atrial fib
  - Lower incidence with MR
- Blood flow in LAA
  - NSR or atrial flutter: velocity > 40 cm/s
  - Atrial fibrillation low velocity flow
- TEE high sensitivity + negative predictive value for LA thrombus

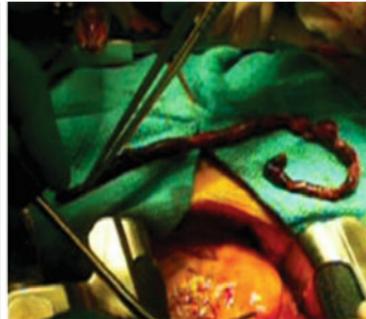
### Pacer Thrombus

Large thrombus encasing a ventricular pacer lead in ME 4C view and during surgery. Surgery photos courtesy of Dr. RJ Cusimano.



### Thrombus in Transit

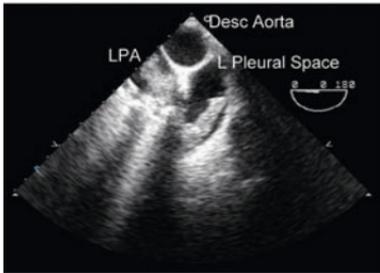
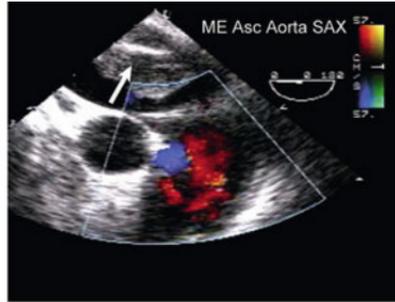
Patient 12 days after radical prostatectomy and lymphadenectomy collapsed at home. A TTE showed an RA thrombus. The ME 4C view shows a wormlike floating right heart thrombus in transit which was removed without requiring CPB.



## Masses Thrombi

### Pulmonary Emboli

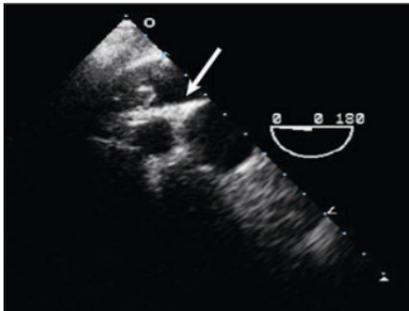
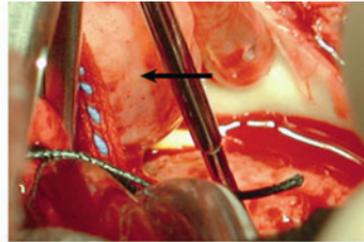
- Risk: stasis + hypercoagulable + intimal damage
- Diagnostic test: helical CT, MRI, angio
- TEE better than TTE, large central
- 2D direct visualization
  - RPA > central > LPA
  - Epicardial exam
- Indirect signs:
  - RV dysfunction
  - TR (mod–severe)
  - IAS bows to the left



### Pulmonary Thomboendarterectomy

Patient with chronic pulmonary emboli and pulmonary hypertension underwent this procedure under circulatory arrest to remove the emboli. (A) ME view of the distal right pulmonary artery (RPA) shows the emboli and (B) at surgery (arrows). The total amount of emboli removed from the pulmonary arteries is shown.

Photos courtesy of Dr Marc de Perrot.



# Endocarditis Vegetations

## Infective Endocarditis

Microbial infection of endocardial heart surface.

3–20% incidence depends on population (native vs prosthetic valves)

### Duke Criteria (Source: Durack DT, et al. Am J Med 1994;96:200-9.)

Pathologic criteria: micro-organisms in vegetations

Clinical criteria: 2 major or 1 major + 3 minor or 5 minor

Major: 1) blood cultures

2) echo findings

- Vegetations: thickened leaflets, mobile masses move through the valve during a cardiac cycle
- New partial valve dehiscence
- New valvular regurgitation

Minor: 1) predisposition (see below), 2) fever, 3) vascular, 4) immunologic, 5) microbiologic, 6) echo findings

- Valve perforations: jet through leaflet, eccentric
- Nodular thickening
- Non-mobile mass

### Predisposition for Endocarditis (Source: Circulation 2007; 116:1736-54.)

#### High risk (use antibiotics)

- Prosthetic valve or repair
- Previous endocarditis
- Heart transplant with cardiac valvulopathy
- Congenital heart
  - Uncorrected cyanotic
  - Repair prosthetic material within 6 months
  - Repair with residua at site of prosthetic material

#### Moderate risk<sup>a</sup>

- Acquired valve
  - Rheumatic disease
  - Degenerative disease
  - MVP with/out MR
- Congenital heart
  - Post-repair ASD, VSD, PDA after 6 months
  - Complex heart defects
- HOCM

#### Low risk<sup>a</sup>

- ASD (isolated)
- Atheroma
- CABG
- Pacemakers

<sup>a</sup>Antibiotics are no longer recommended

### Complications of Endocarditis

- Heart failure: greatest predictor of mortality
- Embolization: mitral > aortic vegetations
- Abscess: hypoechoic area in adjacent tissue without communication with cardiac chamber or vessel, nonpulsatile, no color Doppler flow
- Fistula: abnormal communication between chambers, seen with color Doppler flow
- Pseudoaneurysm of intervalvular fibrosa: echo free area between aortic annulus and base of AMVL, pulsatile with systolic flow from LVOT

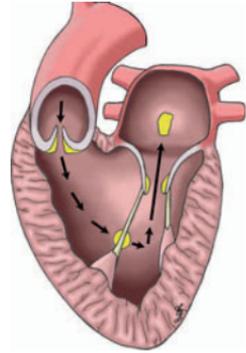
### What to tell the surgeon

- Vegetations (location, size, number)
- Valve pathology (pre-existing)
- Valve function (obstruction, regurgitation)
- Complications (abscess, pseudoaneurysm, fistula)

## Endocarditis Vegetations

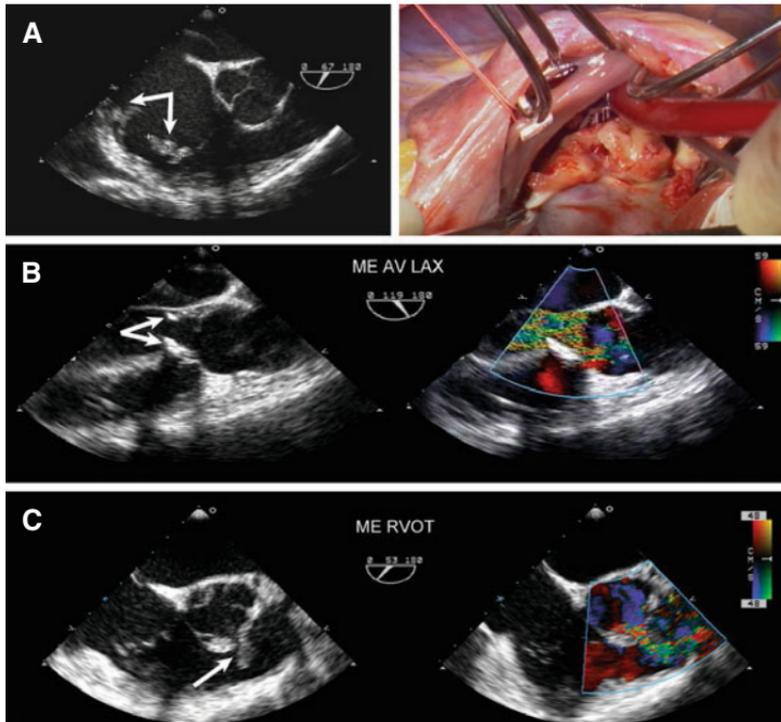
### Vegetations

- Soft tissue density echo
- Irregular shape, size
- Mobile, independent of underlying cardiac structure
- AV > MV > TV > PV, check all valves
- Low pressure side of regurgitant jet:
  - AI jet → LV side AV, chordae MV
  - MR jet → LA side MV, LA wall
  - TR jet → RA side TV
  - VSD orifice → orifice facing RV, 2° on PV and TV
- Obstruction of normal valve function
- Incompetent valve function
- Implanted material



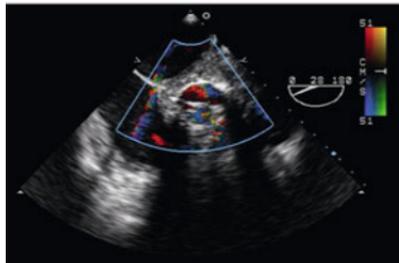
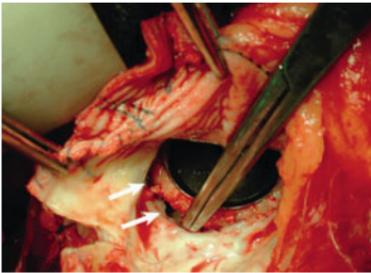
Source: Baddour L, et al. *Circulation* 2005;111:e394-e434.

- (A) Tricuspid valve vegetations shown in RVOT view and at the time of surgery.  
 (B) Aortic valve vegetations (arrow) with severe AI on color flow in ME AV LAX view.  
 (C) Patient with a vegetation (arrow) prolapsing through a perimembranous VSD.

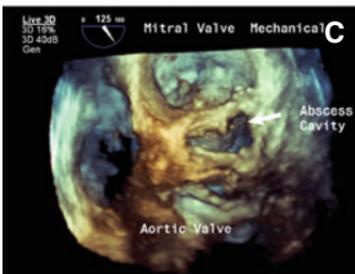
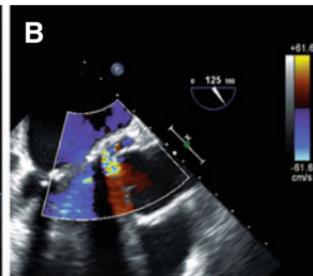
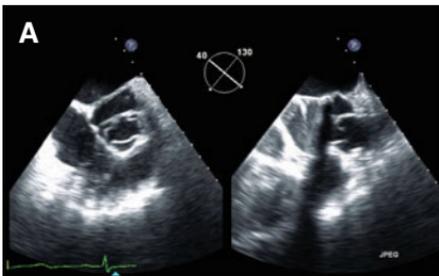


## Endocarditis Complications

**Abscess** is a pus cavity that may involve the annulus, myocardium, or intervalvular fibrosa. It is seen as an echo dense or echo-lucent area around the annulus. Typically, there should be no color flow as shown in this patient with a ST Jude AVR and para-aortic abscess. Below is a picture of the operative finding.



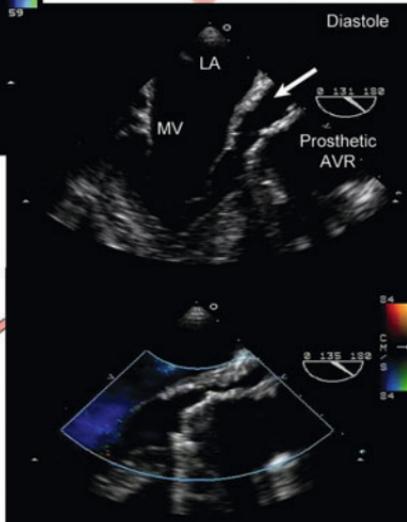
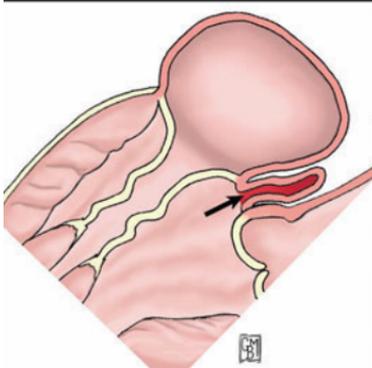
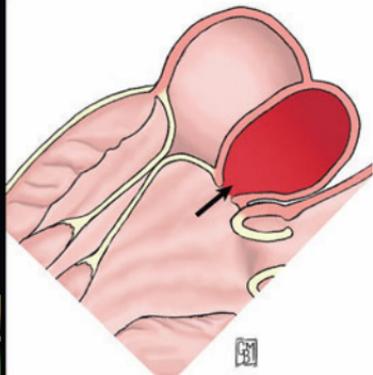
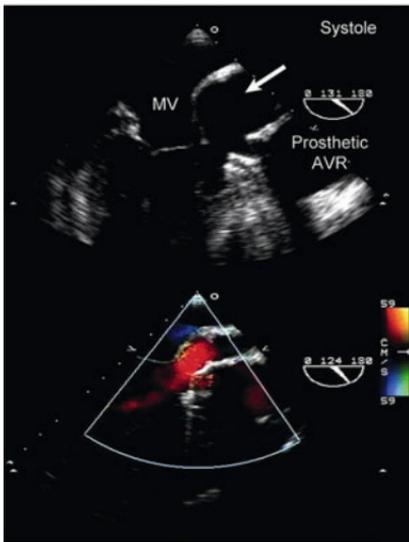
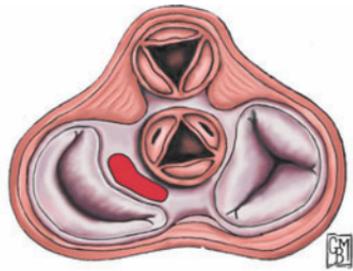
**Abscess** of the intervalvular fibrosa is an echo free area between aortic annulus and LA in a patient with a mechanical MVR. Compare (A) 2D ME AV SAX and LAX (B) color Doppler AV LAX, (C) 3D ME AV SAX views and the (D) intraoperative findings.



## Endocarditis Complications

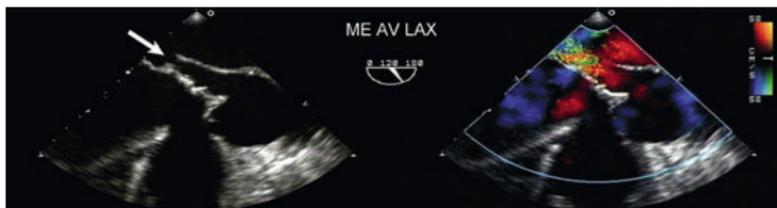
### Pseudoaneurysm

Intervalvular fibrosa pseudoaneurysm (arrow) is an echo free area between aortic annulus and AMVL base. The space is dynamic expanding during systole and is smaller during diastole. Color flow shows early systolic flow and early diastolic emptying.



## Endocarditis Complications

**Fistula** is an abnormal connection between two cavities as a result of an abscess or pseudoaneurysm rupture. Can be identified with color Doppler. Shown here is a fistula (arrow) between the aorta and LA.



**Abscess** of the anterior MV leaflet (arrow) has echo-lucent cavities with a perforation and central MR with color Doppler.



**Dehiscence** of a prosthetic valve presents as an abnormal rocking motion independent of surrounding structures. A gap between the tissue and sewing ring is present with significant paravalvular regurgitation, a dehisced MV ring (arrow) with severe MR.



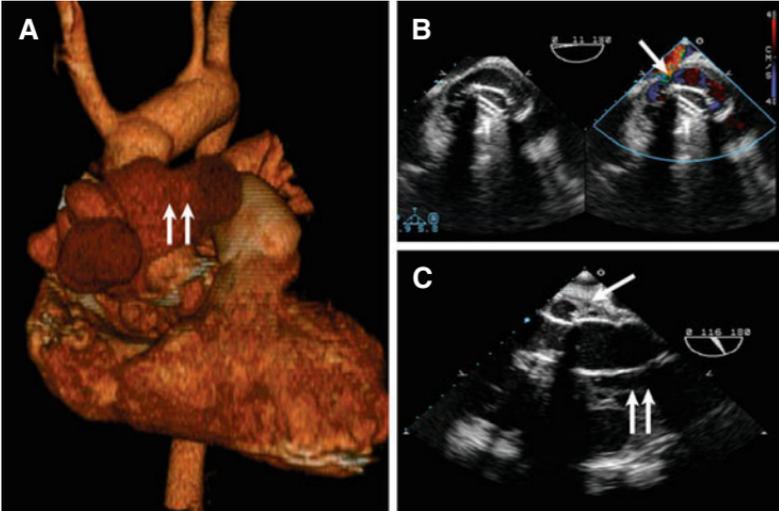
**“Jet lesion”** is a mycotic aneurysm of the AMVL, with a “wind sock” appearance (arrow), that can perforate resulting in MR. This results from impact on the AMVL of the aortic insufficiency jet usually from AV endocarditis.



# Endocarditis Complications

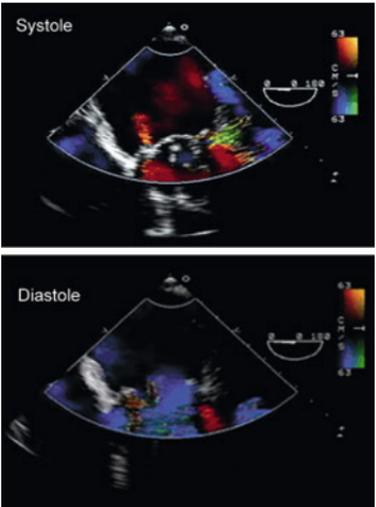
## Pseudoaneurysm to LA Fistula

Patient after Bentall procedure developed a pulsatile suprasternal mass that on (A) CT reconstruction shows an anterior pseudoaneurysm (double arrows) and a (B) posterior pseudoaneurysm (arrow) that has ruptured into the LA creating a fistula. (C) Both are seen in ME AV LAX view, anterior (double arrow) and posterior (arrow).



## AV Groove Separation

This involves disruption of the mitral annulus from the LV resulting in flow outside the heart. This is evident by profuse bleeding in the surgical field. ME 4C view shows systolic flow (green) outside the heart as seen in this patient during mechanical MVR implantation. Treatment includes removing the prosthesis and patch repair to restore continuity.



# Ventricular Assist Devices and Heart Transplant

VADs Overview .....	200–201
VADs Function.....	202–203
VADs Complication .....	204
RVAD and Percutaneous VAD.....	205
Heart Transplantation .....	206–207

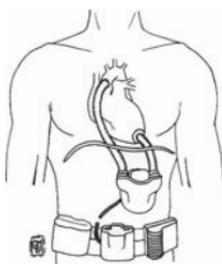
## VADs Overview

### Ventricular Assist Devices (LVAD, RVAD)

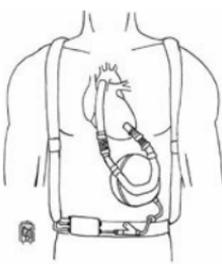
Mechanical ventricular assist devices are used to support the LV (LVAD), RV (RVAD), or both ventricles (BiVAD). They rely on an inflow cannula into the device (outflow from patient) typically placed in the supported ventricle (RVAD or LVAD) or atrium (RA for RVAD). The outflow cannula from the device (inflow into the patient) is placed in the aorta (LVAD) or PA (RVAD).

### Pulsatile VADs

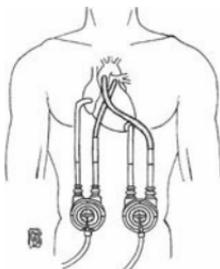
The pump with valves provides asynchronous (to the native heart) positive displacement of blood into the patient's systemic circulation.



Implanted LVAD: cannulae and device both internal. Power source cable external.



Extra corporeal LVAD: internal cannulae join external device and power cable.



Biventricular support system: internal cannulae join external devices and power cables.

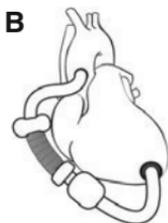
### Continuous Flow VADs

These devices have replaced pulsatile VADs as the favored devices for bridge to transplant and destination therapy. These are axial flow devices that use a propeller screw type design rotating at rapid rates to push blood continuously forward. They are small, totally implantable and durable with a simple valveless design. The DeBakey VAD and HeartMate II (HM II) use typical inflow and outflow cannulae with the axial pump implanted in the thorax. The impeller in the Jarvik 2000 device is implanted directly in the LV apex with the outflow conduit in the descending aorta (left thoracotomy) or ascending aorta (sternotomy). The DeBakey and Jarvik devices can support either the R, L, or both ventricles, while the HM II only the LV.

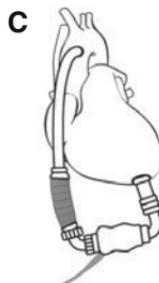
Images permission of Frances Yeung



Jarvik 2000



DeBakey VAD



HeartMate II

## VADs Overview

### Ventricular Assist Devices (LVAD, RVAD)

TEE SCA category 2 indication

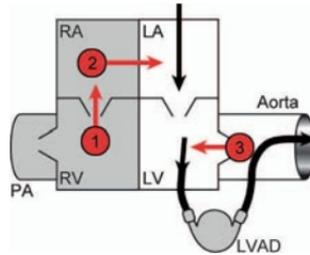
Pre-CPB

Absolute indications:

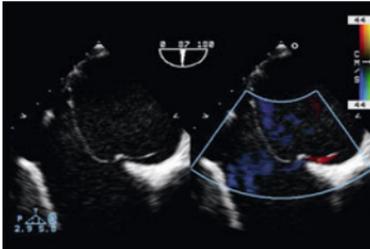
1. LV and RV function and size
  - RV function determines LVAD filling
2. PFO or ASD
  - Post-LVAD hypoxemia R→L shunt
  - Paradoxical emboli
3. Aortic Insufficiency
  - LVAD loop→poor systemic perfusion

Preexisting conditions:

- Intracavitary thrombus
- Aortic atheroma
- Tricuspid regurgitation
- Mitral regurgitation/stenosis

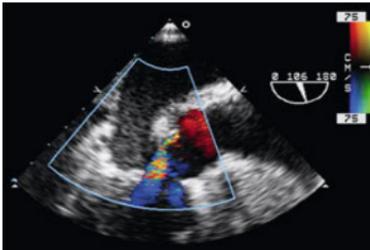


Source: Chumnanvej S, et al. *Anesth Analg* 2007;106:583-401.



### Patent Foramen Ovale (PFO)

- May be difficult to detect as:
  - LAP > RAP
  - Septum bowed to right/immobile
- Valsalva will increase RAP
  - Color Doppler ± Valsalva
  - Bubble study ± Valsalva
- If PFO present needs to be closed
- Recheck for PFO post-CPB



### Aortic Insufficiency (AI)

- Underestimate AI severity pre-CPB as have reduced transaortic valve gradient from low aortic pressure – high LVEDP
- Can check AI on CPB (shown) as have high aortic pressure like LVAD flow
- LV vent drain > 1.5 L/min is significant
- Repair or replace AV if moderate to severe AI



### Intracavitary Thrombus

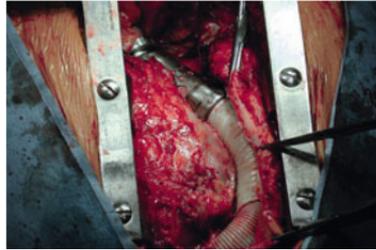
- Smoke indicates low flow in:
  - Ventricles, atria, aorta
- LAA clot: tie off LAA
- LV clot (arrow): carefully remove as
  - May occlude cannula
  - May embolize

# VADs Function

## Ventricular Assist Devices (LVAD, RVAD)

TEE SCA category 2 indication  
Post-CPB

- Deairing
  - Reduced size
  - Interventricular septum position
  - AV may or may not open
- RV function (TR severity, RVSP)
- Recheck PFO
- Aortic insufficiency
- Device function
  - LV conduit and gradient
  - Aorta conduit and gradient

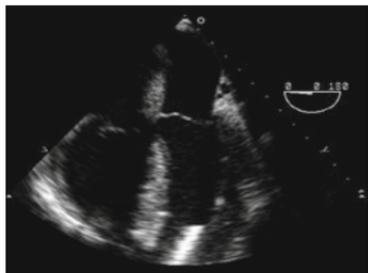


Source: Chumnanvej S et al.  
Anesth Analg 2007;106:583-401.



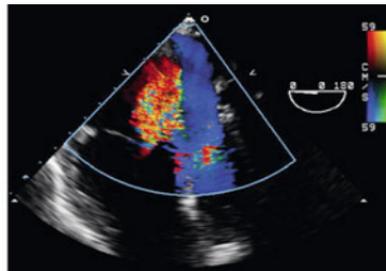
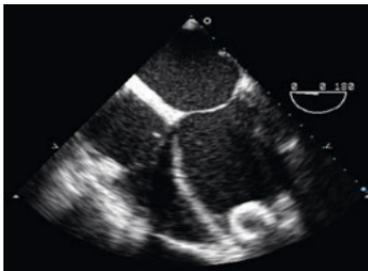
### Device Deairing

- Device is started on CPB
- Air in ascending aorta, proximal to aortic conduit
- Air may traverse the RCA further impairing RV function
- If continuous air consider air entrainment through open suture line or cannula displacement

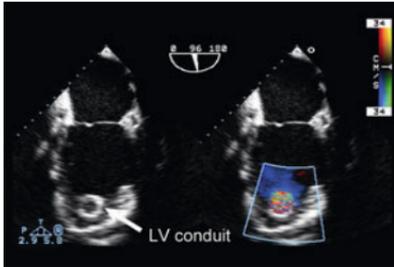


### LV Decompression

- Pulsatile devices nearly empty the LV
- Continuous flow devices partly empty LV
- Septal position important for RV function
  - Left bowed: too decompressed LV
  - Right bowed: not decompressed LV
  - Neutral: best position optimizes RV
- RV function may:
  - Worsen with  $\uparrow$  preload
  - Improve  $\downarrow$  afterload

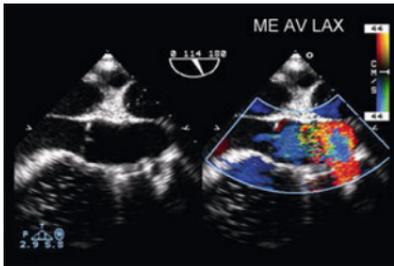
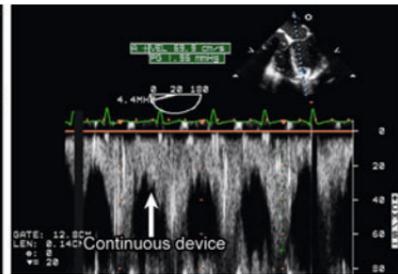
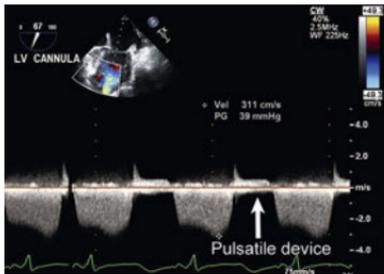


## VADs Function



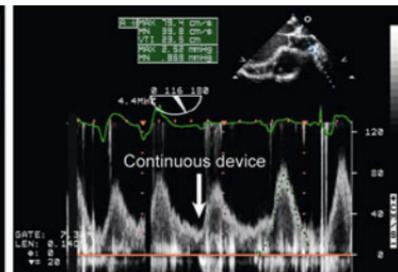
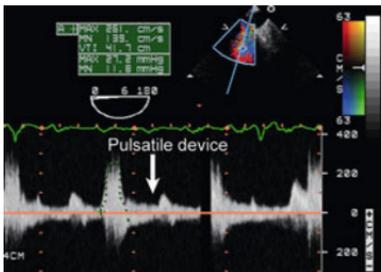
### LV Apical Cannula

- Device inflow, patient outflow
- Positioned away from IVS + LV walls, towards MV, seen in two orthogonal views
- Color: laminar unidirectional flow
- Spectral Doppler (PW or CW):
  - Pulsatile: discrete, < 2.3 m/s
  - Continuous: not to baseline (arrow) 1.0–2.0 m/s



### Aortic Cannula

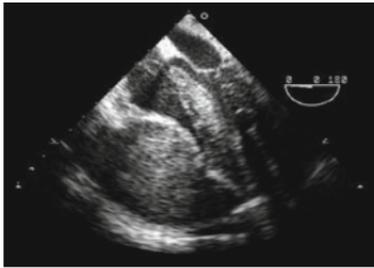
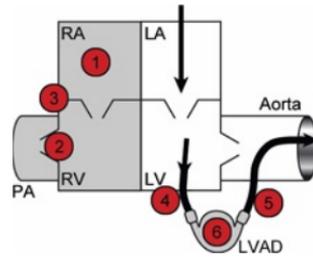
- Device outflow, patient inflow
- Positioned antero-lateral ascending aorta, angulated, pull TEE probe back
- Color: turbulent unidirectional flow
  - Assess aortic insufficiency (AI)
- Spectral Doppler (PW or CW):
  - Pulsatile: discrete, 2.1 m/s, asynchronous to ECG
  - Continuous: not to baseline (arrow) 1.0–2.0 m/s, pulsatile pattern is from LV contraction synchronous with ECG



## VADs Complication

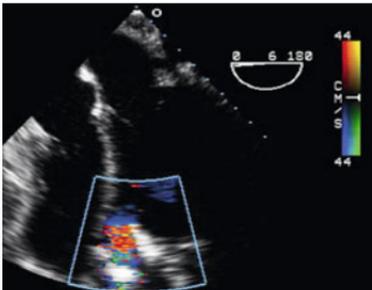
### VAD Complications

- Device low output
  1. Hypovolemia (empty RV)
  2. RV failure (dilated, hypofunction, TR)
  3. Cardiac tamponade
  4. Inflow cannula obstruction
  5. Outflow cannula obstruction
  6. Device valve failure
- Device high output
  - Sepsis
  - Aortic insufficiency
- Thrombus
- Hypoxemia
- Device failure



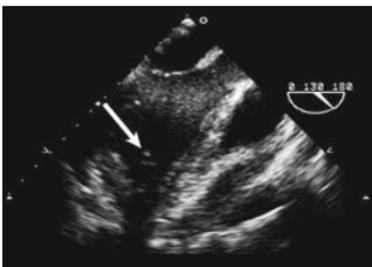
### Cardiac Tamponade

- Local or circumferential pericardial effusion
- Chamber compression: RA, RV (shown)
- Compromise LVAD filling
- Common early or late problem as patients may require anticoagulation post-operatively and be prone to bleeding.
- Require drainage of fluid or evacuation of hematoma



### Inflow Cannula Obstruction

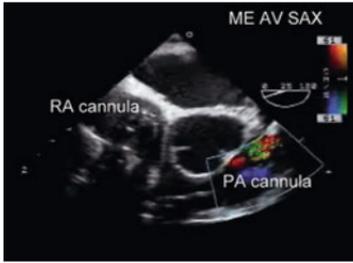
- Etiology of obstructed orifice:
  - Malposition towards LV wall (shown)
  - Hypovolemia resulting in chamber collapse around cannula
  - Thrombus occluding cannula
- Doppler
  - Color: turbulent flow
  - Spectral: velocity > 2.3 m/s
- Results in device low flow



### Cannula Thrombus

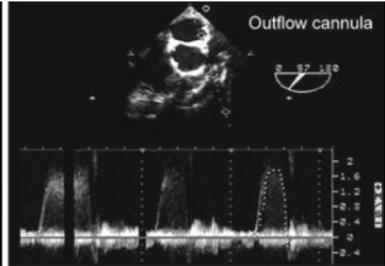
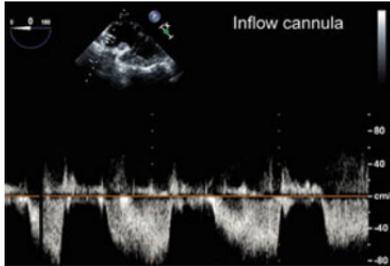
- Thrombi may form in stagnant areas of the cardiac chambers.
- Cannula thrombus may also occur and result in obstruction to cannula flow and device low flow. Shown is a small thrombus on tip of LV cannula (arrow).
- Anticoagulation is often required to prevent thrombus formation.

## RVAD and Percutaneous VAD



### Right Ventricular Assist Device

- Indicated for severe RV dysfunction
- Pulsatile or continuous flow device
- Device inflow cannula: RA or RV
  - Discrete unidirectional flow
  - Velocity 1.0–2.0 m/s
- Device outflow cannula: PA
  - Discrete unidirectional flow
  - Velocity 1.0–2.0 m/s



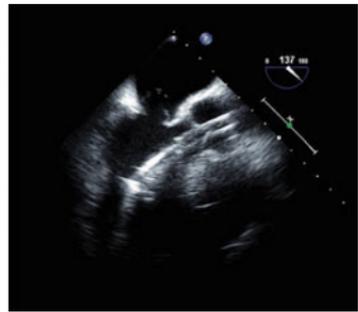
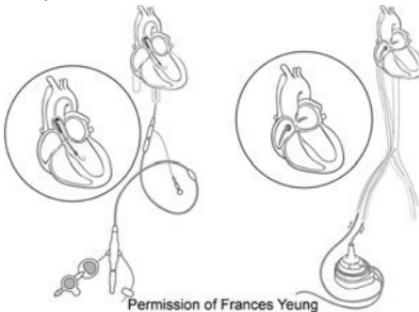
### Percutaneous Ventricular Assist Device

Percutaneous VADs provide temporary (5–14 days) partial or total circulatory support. These devices are inserted through the femoral artery and continuously recirculate oxygenated blood from the left heart into the systemic circulation.

- The Impella Recover LP system is a ventricular unloading catheter that aspirates blood from the LV and expels it into the ascending aorta using a microaxial flow blood pump. The 12F catheter is inserted retrograde via a femoral artery and is positioned across the AV. On TEE the inflow port of the catheter is in the LVOT 3–4 cm from the AV. The outflow port is 1.5–2.0 cm distal to the sinus of Valsalva.
- The TandemHeart percutaneous LVAD is a LA to femoral artery system with three parts. A 21F femoral venous cannula is inserted into the RA and directed transseptally into the LA under fluoroscopy or TEE guidance. This cannula is attached as inflow to an extracorporeal centrifugal pump. Outflow from the pump is a 15–17F catheter inserted in the right femoral artery to the aortic bifurcation.

Impella recover

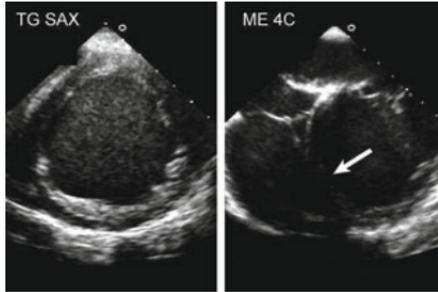
TandemHeart



# Heart Transplantation

## TEE in Heart Failure

- Dilated ventricles (R < L)
  - EDD > 80 mm
  - ESD > 55 mm
- Thinned myocardium
  - Thickness < 6 mm
- Low cardiac output
  - Wall motion, global
  - Smoke
  - Quantify
- Mitral regurgitation
- Tricuspid regurgitation
  - Estimate systolic PAP



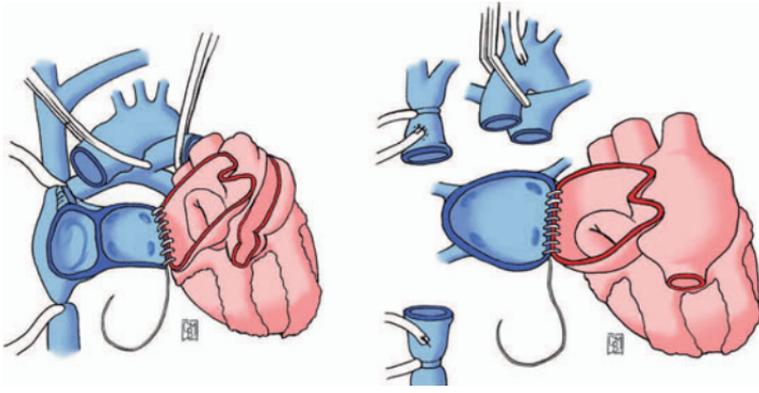
## Heart Transplant Technique

### Bi-atrial (Lower-Shumway)

Part of recipient RA, LA, and pulmonary veins (blue) are preserved and sutured to donor RA and LA (red).

### Bicaval (Wythenshawe)

Recipient native RA is removed and anastomosis is between donor (red) and native (blue) SVC, IVC and LA.



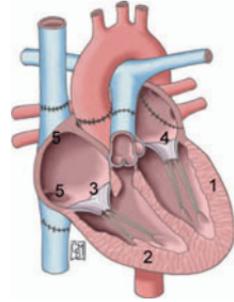
## Post-CPB Findings (SCA category 2 Indication)

- Deairing
- Ventricular function
  - Global
  - Regional
- Tricuspid regurgitation (estimate PASP)
- Anastomotic sites
  - SVC
  - IVC
  - LA
- PFO

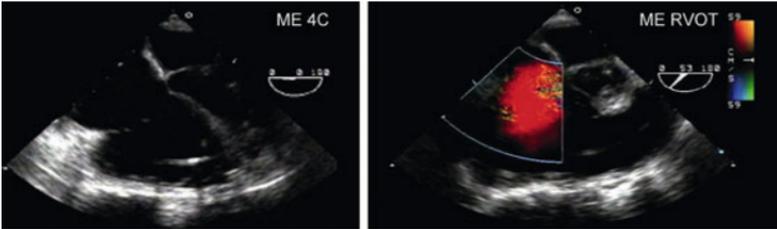
## Heart Transplantation

### Acute Complications of Heart Transplant

1. LV failure (global, regional)
  - Dilated
  - Wall motion abnormal
  - IVS septal bowing
2. RV failure
  - Biventricular failure: ? early rejection
  - Anastomotic sites
3. TR severe (also RVSP)
  - Anastomotic sites
4. LA / MV inflow obstruction
5. SVC, IVC obstruction

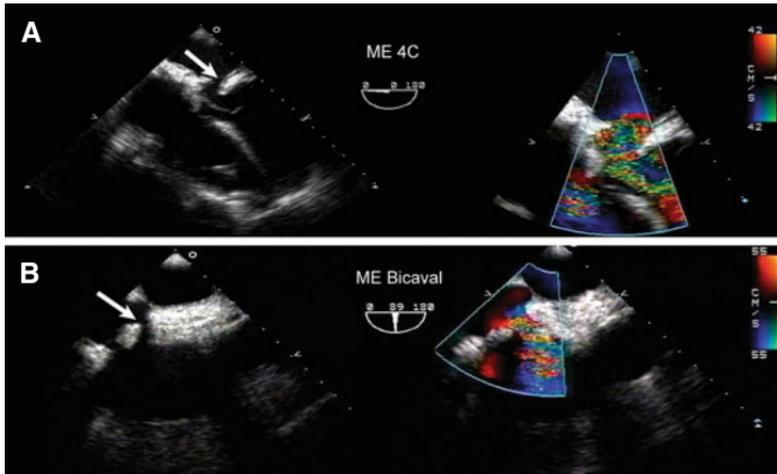


Assess RV function as it is commonly decreased post heart transplant from pre-existing elevated PAP. The RV shown here is dilated with the IVS bowed into the LV. Significant TR may be present, laminar TR indicates severe RV dysfunction.



(A) LA anastomotic stenosis (arrow) with turbulent flow above the mitral valve restricting LV filling.

(B) IVC stenosis (arrow) with turbulent flow that required postoperative stenting.



# Hypertrophic Obstructive Cardiomyopathy and Diastolic Dysfunction

HOCM Overview .....	210-211
HOCM TEE Findings.....	212-213
Diastolic Function .....	214-215
Diastolic Function TEE Assessment.....	216-217

## HOCM Overview

### Clinical

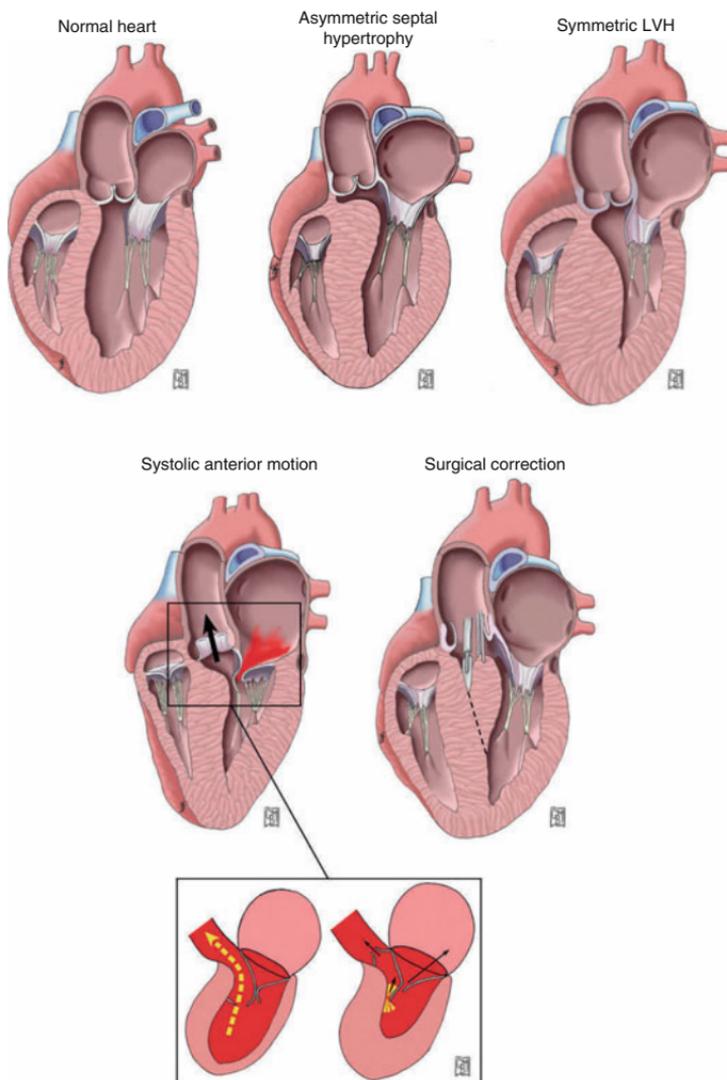
- The left ventricle can have symmetric or asymmetric hypertrophy.
- Asymmetric septal hypertrophy has unexplained LV hypertrophy, with LV obstruction (LVOT or mid-cavitary) and abnormal diastolic function.
- Familial autosomal dominant with variable penetrance, related to abnormalities of the B myosin heavy chain. Incidence of 1:500.
- Usually asymptomatic though presenting symptoms include LVOT obstruction (syncope, sudden death), myocardial ischemia (angina) and diastolic dysfunction (pulmonary congestion and shortness of breath).
- Surgery involves a transaortic septal myectomy with a parallel incision of the septum below the right coronary cusp to the papillary muscles. Postoperative complications include complete heart block (10%), VSD (0.6%), LV rupture (1%), dysrhythmias (A = 26% and V = 7.3%).

Differential asymmetrical hypertrophy	Differential symmetrical hypertrophy
Familial	HBP
Sigmoid shaped in elderly	Aortic stenosis
Apical variant	Infiltration (amyloid, glycogen, sarcoid)
Eisenmenger's	Metabolic (Cushing, diabetes)
Septal sarcomas	Renal disease
LVH with lateral wall infarct	Athletic heart, obesity
Pulmonary hypertension + RVH	Congenital (Fabray, Noonan, Friedrich's Ataxia)
HBP, hemodialysis	

### Pathophysiology

- In the normal heart, the LVOT is formed by the IVS and AMVL. In HCM, there is asymmetrical hypertrophy of LV septum with sparing of posterior basal LV wall.
- In patients with septal hypertrophy, the LVOT is narrowed. The papillary muscles are anteriorly displaced and the MV leaflets elongated. During systole, the body of the AMVL coapts with the tip of the PMVL. The tip of the AMVL is dragged into the LVOT by the Venturi effect of systolic flow, termed systolic anterior motion (SAM). The AMVL contacts the septum at the septal contact point.
  - For every LVOT diameter, there is a critical velocity threshold above which the Venturi effect is established.
  - Results in dynamic late peaking (dagger) high velocity systolic gradient.
  - Assess LVOT gradient clinically at rest and post-PVC ( $\uparrow$  contractility), Valsalva ( $\downarrow$  preload), and amyl nitrate ( $\downarrow$  preload,  $\downarrow$  afterload).
  - Obstruction is worsened by low intravascular volume, hypercontractility and low afterload.
  - Obstruction is improved by increased intravascular volume, decreasing contractility, and increasing afterload.
- The displaced AMVL results in poor MV leaflet coaptation and eccentric posterior directed MR in mid to late systole.
- Normal LV systolic function with impaired diastolic function.
- SAM may occur in patients with symmetric LVH from aortic stenosis or HBP post-AVR or post-MV repair in patients with an anterior displaced papillary muscle and elongated MV leaflet.

## HOCM Overview



Systolic anterior motion (SAM) of the mitral valve leaflets in early and late systole as a result of the Venturi effect the AMVL may contact the IVS. Leaflet malcoaptation leads to a posterior directed MR jet.

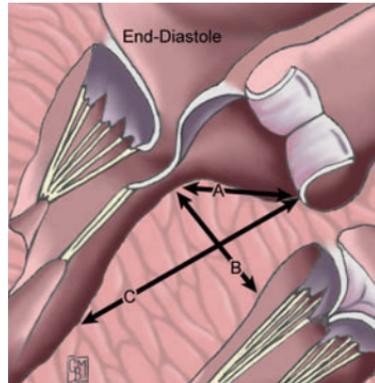
## HOCM TEE Findings

### Hypertrophic Obstructive Cardiomyopathy (HOCM)

#### TEE Findings Pre-CPB

##### 2D Imaging

- LV wall thickness (septal, lateral), symmetric or asymmetrical  
Septal:free wall ratio > 1.3:1  
> 15 mm thickness is abnormal
- Diameter LVOT
- Mitral valve:
  - No intrinsic MV disease
  - SAM of anterior MV leaflet (AMVL)
- Septal contact point with IVS: measure
  - A. Distance RCC to septal contact point
  - B. Septal thickness
  - C. Distal point of septal thickening
- Anomalous papillary muscle insertion directly into the leaflet, 10% of patients
- ↑ LA size > 40 mm or > 20 cm<sup>2</sup> in 4C
- LV and RV systolic function, normal or hyperdynamic



##### Color Doppler

- Turbulent LVOT flow
- Mitral regurgitation, eccentric posterior directed jet

##### Spectral Doppler

- PW point of peak gradient (mid-cavitary or LVOT)
- CW peak and mean LVOT gradient:
  - Late peaking systolic flow (dagger shaped)
  - Gradient increases post-PVC and with amyl nitrate
- MV inflow: LV diastolic dysfunction
- Pulmonary vein pattern abnormalities consistent with diastolic dysfunction

#### TEE Findings Post-CPB

##### 2D Imaging

- Measure residual septal thickness
- SAM and residual MR (with adequate ventricular filling and BP)
- LV and RV systolic function (LAD muscle bridge)

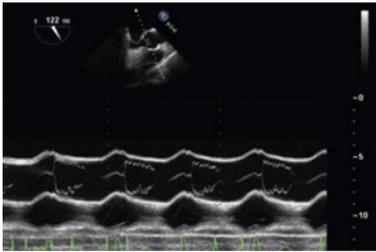
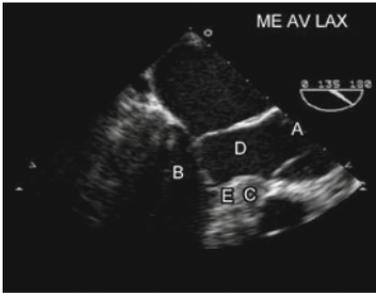
##### Color Doppler

- Laminar systolic LVOT flow
- MR from intrinsic valve disease
- VSD (< 3 mm IVS) absent, high velocity L to R flow in systole and diastole
- Septal perforator flow into LV during diastole

##### Spectral Doppler

- CW peak LVOT gradients (at rest and post-PVC)
- Mid-ventricular cavitary obstruction
- MV inflow diastolic function
- Pulmonary vein flow

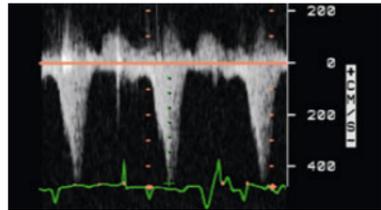
## HOCM TEE Findings



### Pre-myectomy 2D Image (ME AV LAX)

- A. During systole AV open
- B. SAM of AMVL tip into LVOT
- C. Septal thickness > 15 mm (EDD)
- D. LVOT diameter
- E. Septal contact point

An M-mode through the aortic valve shows fluttering of the AV cusps.



### Color Doppler

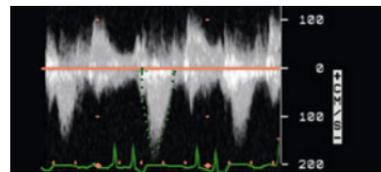
- MR eccentric posterior directed
- Turbulent LVOT flow below AV

### Spectral Doppler

- CW: late peaking LVOT gradient (dagger shaped)
- Peak instantaneous gradient > 36 mmHg is significant LVOT obstruction, often higher than at heart catheterization
- Post-PVC gradient is even higher
- PW in ME views for peak gradient from LV intracavitary obstruction

### Post-myectomy

- Septal thickness
- LVOT diameter
- Residual SAM
- Residual MR
- Peak gradient LVOT
- VSD absent

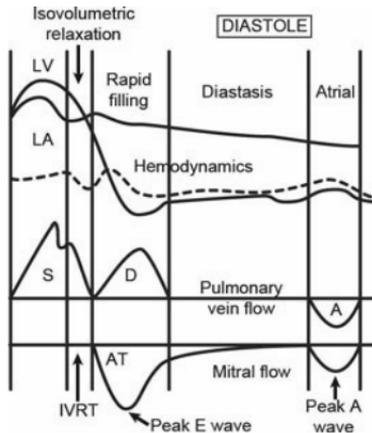


# Diastolic Function

## Left Ventricular Diastolic Function

### Four Phases of Diastole

1. Isovolumetric Relaxation (IVRT): closure AV to open of MV, LV Pressure falls
2. Rapid ventricular filling (**E wave velocity**): MV opens, accumulating blood from LA enters LV increasing LV Pressure
3. Diastasis (**slope of filling**): LAP and LV Pressure equalize so no flow despite semi-open MV leaflets a slower filling period.
4. Atrial contraction (**A wave velocity**): atrial contraction, LAP > LV Pressure allows MV to reopen wider to fill LV, contributes 15–20% of LV preload

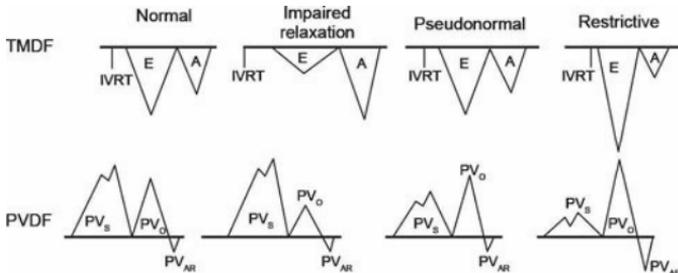


### Four Phases of Diastole

1. Isovolumetric relaxation time (IVRT)
  2. peak "E" and "A" wave velocities, (E =  $0.8 \pm 0.2$  m/s, A =  $0.5 \pm 0.2$  m/s)
  3. "E" wave deceleration time (DT) from peak to baseline
  4. "E" (TVI<sub>E</sub>) or "A" (TVI<sub>A</sub>) wave velocities (n > 8 cm/s, DD < 8 cm/s)
  5. Ratio of "E" and "A" velocities (E:A ratio)
  6. Ratio of pulmonary (A<sub>p</sub>) and mitral inflow (A<sub>m</sub>) "A" wave duration
  7. Propagation velocity (V<sub>p</sub>) n > 55 cm/s, DD < 50 cm/s
- n = normal, DD = diastolic dysfunction

### Phases of Diastolic Dysfunction

Parameter	Normal	Impaired relaxation	Pseudonormal filling	Restrictive filling
E Wave DT (ms)	160–240	> 240	160–200	< 160
IVRT (ms)	70–90	> 90	< 90	< 70
E : A	1–2	< 1	1–1.5	> 1.5
A <sub>m</sub> : A <sub>p</sub> Duration	A <sub>m</sub> <sup>3</sup> A <sub>p</sub>	A <sub>m</sub> > A <sub>p</sub>	A <sub>m</sub> < A <sub>p</sub>	A <sub>m</sub> << A <sub>p</sub>
PV <sub>s</sub> : PV <sub>D</sub>	PV <sub>s</sub> > PV <sub>D</sub>	PV <sub>s</sub> > PV <sub>D</sub>	PV <sub>s</sub> < PV <sub>D</sub>	PV <sub>s</sub> << PV <sub>D</sub>



## Diastolic Function

- Diastolic dysfunction is a limitation of the ventricle to fill to normal end-diastolic volume (EDV) without an abnormal increase in end-diastolic pressure (EDP) at rest or during exercise.
- Diastolic heart failure is defined as a condition in which filling of the LV is impeded resulting in symptoms of low cardiac output, elevated LV filling pressures, or both.
- Common causes of diastolic dysfunction include: HBP, CAD, restrictive and dilated cardiomyopathies, constrictive pericarditis
- Occurs in 30–75% of cardiac surgical patients
- Increases morbidity, difficulty weaning from CPB and need for inotropic support.

Diastolic dysfunction can manifest as the following patterns:

- **Impaired relaxation:** This pattern is seen in patients with reduced LV relaxation rate but relatively normal compliance and filling pressures. This occurs in acute MI or ischemia, LVH, HCM, inhalation anesthetics, prolonged CPB. Diagnosis includes an E/A ratio  $< 1$ , prolonged DT  $> 220$  msec, IVRT  $> 100$  msec, S/D  $> 1$  and prominent AR. Color M-mode Vp is reduced  $< 45$  cm/s, as well as DTE Em  $< 8$  cm/s, prolonged E-wave DT, an E:A ratio of  $< 1.0$ , a PVs  $\gg$  PVD wave, and a AM duration  $> Ap$  duration.
- **Pseudonormal:** This is a combined pattern of abnormal relaxation and restrictive physiology. As a result of abnormal relaxation, LA pressures increase to compensate with a normalization of the mitral inflow E and A wave. This occurs with end stage cardiac disease. Standard Doppler indices are characterized by high E, E/A ratio  $< 1$ , IVRT  $< 100$  ms, DT  $< 220$  ms, S/D  $> 1$  and prominent AR. Since LV relaxation is impaired color M-mode Vp remains reduced,  $< 45$  cm/s, as well as DTE Em  $< 8$  cm/s.
- **Restrictive:** This pattern has profound abnormalities of LV relaxation, compliance and markedly increased filling pressure. Such disease processes would include advanced ischemia heart disease, uncompensated congestive failure, or restrictive cardiomyopathy. Standard Doppler filling indices are characterized by an increased E/A ratio  $> 2$ , short DT  $< 150$  ms, IVRT  $< 60$  ms, S/D  $< 1$ . AR may be normal or small due to atrial mechanical failure. Color M-mode Vp and DTI Em are the lowest. The E:A ratio decreases with reduction of preload or positive pressure ventilation. With the administration of nitrates, diuretics, or positive pressure ventilation, Stage 3 physiologic pattern of dysfunction reverts to Stage I dysfunction.
- **Normal filling pattern:** The normal filling pattern is seen in patients with normal LV relaxation rate, compliance and filling pressures. Standard Doppler indices of LV filling and PV flow are characterized by high E, E/A ratio  $< 1$ , IVRT  $< 100$  ms, DT  $< 220$  ms, and S/D  $> 1$ . Children, young adults, and athletes may have very short IVRT, prominent E, very short DT and S/D  $< 1$ . Color M-mode Vp is fast, usually  $> 55$  cm/s in younger and  $> 45$  cm/s in older adults. DTI Em velocity measured in the LV long axis plane is  $> 10$  and  $> 8$  cm/s respectively.
- Can be assessed intraoperatively using 2D, M-mode, PW, and tissue Doppler imaging modes as shown on the following pages. Many of the indices are preload, afterload, HR, and rhythm dependent.

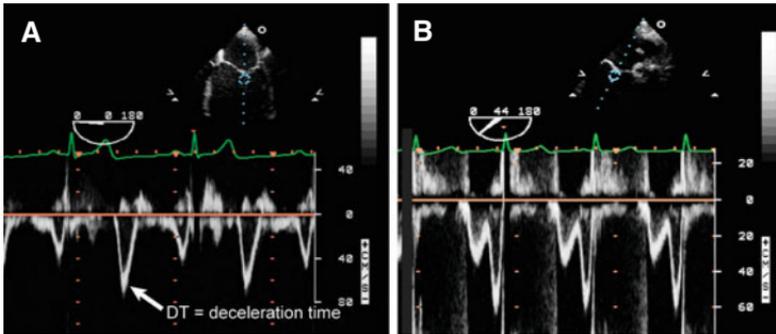
### 2D mode:

- Significant diastolic dysfunction is unlikely in the presence of a structurally normal heart. Common 2D echo findings in diastolic dysfunction include global and regional LV and RV systolic dysfunction, LA enlargement, caval and hepatic dilation, increased LV mass, and pericardial thickness. In addition, 2D echo helps in excluding other causes of heart failure such as valvular heart disease.

## Diastolic Function TEE Assessment

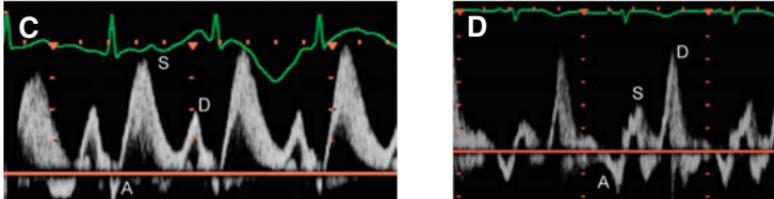
### Transmitral Doppler Flow

ME 4C view (PW at leaflet tips) for mitral inflow profile of early (E) and late (A) velocities and deceleration time (DT). DT is measured by extending the deceleration slope from peak E wave velocity to the baseline. (A) Normal pattern or Pseudo-normal ( $E > A$ ) pattern is distinguished by pulmonary vein flow pattern. (B) The Doppler for impaired relaxation pattern shows  $E < A$  wave.



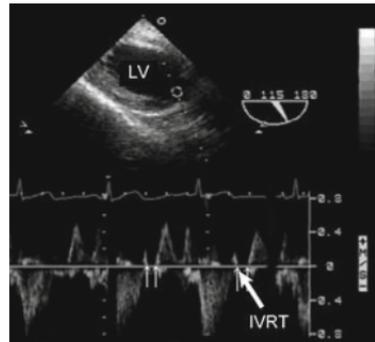
### Pulmonary Vein Flow Doppler

(C) Normal pulmonary vein flow pattern is found with normal diastolic function. (D) A blunted pulmonary vein flow pattern is found in diastolic dysfunction.



### IVRT

Deep TG LAX view for Isovolumic relaxation time (IVRT) with PW placed between the mitral valve inflow and LVOT (maximum gate length). Arrow indicates, the points of closure of aortic valve and opening of mitral valve representing the IVRT measurement.



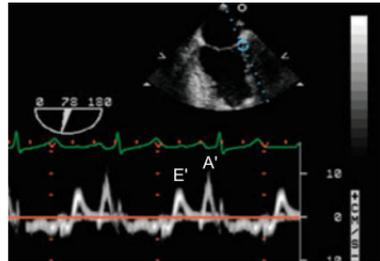
## Diastolic Function TEE Assessment

### Tissue Doppler Imaging (TDI)

- TDI displays the velocities of the myocardium during contraction and relaxation. TDI focuses on the high intensity, low velocity echoes of the myocardium.
- Pattern has a
  - Systolic component
  - Diastolic component (biphasic)
  - Normal  $E' > A'$  (index of relaxation)
  - Abnormal  $E' < A'$ , or  $E' < 8$  cm/s (impaired relaxation, see below)
- Clinical studies have shown an inverse relationship between TDI diastolic myocardial velocities and LV relaxation. Preload independent.

To obtain TDI:

- PW mode TDI is preset on some machines
- Use ME 2C or 4C view
- Place the sample volume of 2–5 mm in the myocardial wall often lateral wall, preferably without wall motion abnormalities.
- Adjust the sweep speed 50–100 mm/s

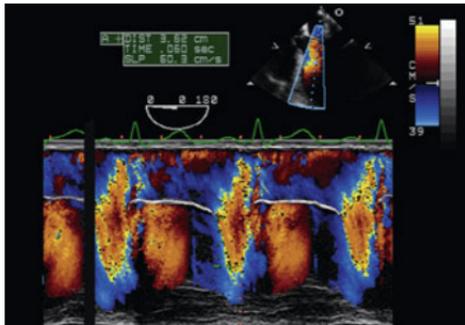


### Color M Mode Transmitral Flow (Vp)

- Color M-mode transmitral flow Doppler visualizes both the temporal and spatial propagation of flow along a single scan line (rather than a single sample volume), over the entire length of the LV during diastole.
- The velocity at which flow propagates within the ventricle ( $V_p$ ) is given by the slope of the color wavefront.
- Color M-mode Doppler  $V_p$  is reduced in ventricles with delayed LV relaxation.

To obtain color M-mode:

- Cursor placed parallel to color mitral inflow jet. Adjust to obtain longest column of color flow from mitral valve to apex.
- Shift Doppler map to alias at 75% of peak E velocity
- Measure slope along a distinct isovelocity (aliasing) line during early filling from the mitral valve plane up to 4 cm into the LV cavity.



# Pericardium

Anatomy .....	220
TEE Views .....	221
Pericardial Effusion .....	222
Cardiac Tamponade .....	223
Pericarditis .....	224-225

## Anatomy

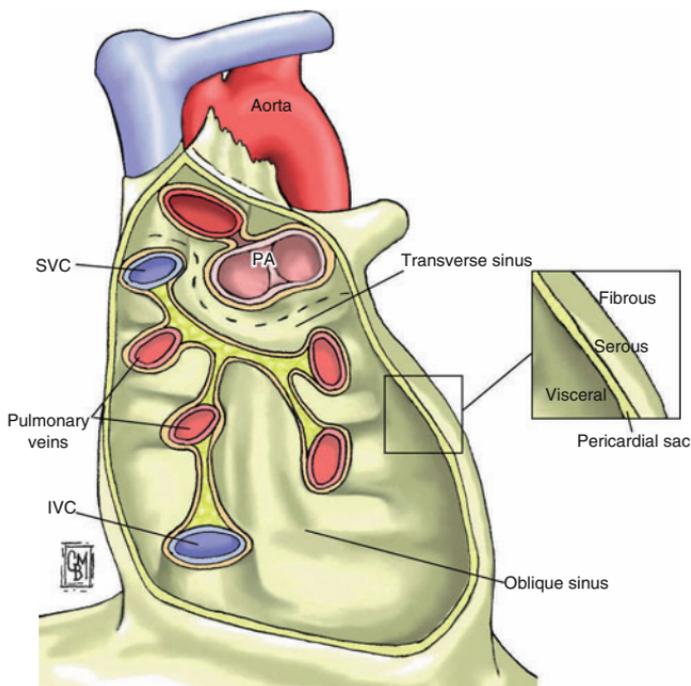
### Pericardial Anatomy

The parietal and visceral pericardium are continuous where the major vessels enter and leave the heart. The parietal pericardium has two inseparable parts, an outer fibrous and inner smooth serous layer. The potential space between the visceral and serous parietal pericardium is the pericardial cavity. Normally 15–50 cc of clear pericardial fluid is in the pericardial sac to reduce friction between pericardial surfaces.

The **oblique sinus** lies behind the left atrium so that the posterior wall of the left atrium is actually separated from the pericardial space. A posterior pericardial effusion behind the LV is often seen behind the left atrium.

The **transverse sinus** is the connection between two tubes of pericardium that envelop the great vessels. The aorta and pulmonary artery are enclosed in one antero-superior tube, and the vena cava and pulmonary veins are enclosed in a more posterior tube. Pericardial effusion located in the superior recess should not be mistaken for an intimal flap of an aortic dissection.

Pathology of the pericardium includes cysts, effusions, thickening, constriction, and tumor. Rarely there can be complete absence of pericardium.



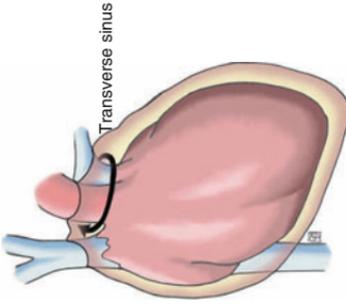
## TEE Views

### Transverse Sinus

Space between pulmonary artery and anterior LA (arrow). RVOT view shows the left atrial appendage (LAA) and a small amount of fluid (arrow) in the transverse sinus without color flow.



### Pericardial Effusion

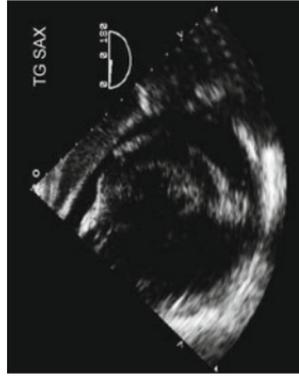


### Oblique Sinus

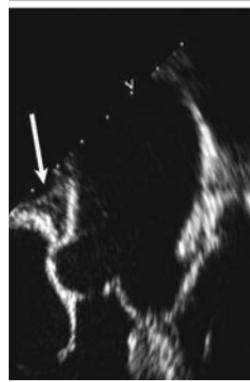
Space between the posterior LA wall and esophagus is filled with fluid compressing the LA. This is visible from most ME views often before weaning from CPB.



TG view images a circumferential pericardial effusion around the entire LV.



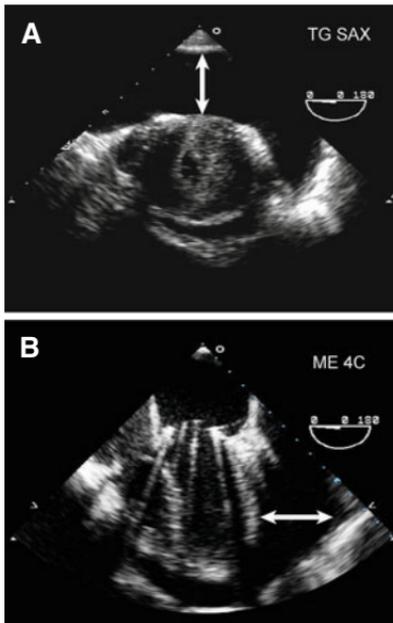
The transverse sinus is shown here at the level of the ascending aorta and right pulmonary artery (RPA) in the ME Aortic Valve and Ascending Aorta LAX views. The space may appear filled with thrombus (arrow) or fluid (arrow).



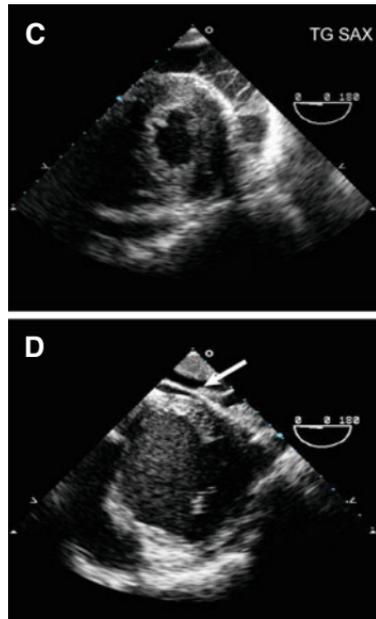
## Pericardial Effusion

### Pericardial Effusion

- Etiology: inflammatory, infectious, neoplastic, post-MI, trauma, or cardiac surgery
- Location (circumferential, loculated)
  - Pericardial effusions surround the heart (4C, bicaval, TG views)
  - Pleural effusions lie posterolateral to descending aorta (desc aortic SAX view)
  - Loculated effusions: post cardiac surgery, inflammatory, metastatic disease
- Echo free (echolucent) stripe between visceral and parietal pericardium
  - Anterior effusion is imaged in ME views, posterior effusion in TG view
  - ↓ echo gain setting to identify pericardial interface (brightest reflector)
  - Isolated anterior echo free space may be an epicardial fat pad
  - Fibrin strands in long-standing effusions or from metastases
  - Hematoma frequently has a similar echo-consistency as myocardium
- Physiological effects depend on rate and volume of accumulation



Size: Small < 1.0 cm (< 200 cc)  
 Moderate 1–2 cm (200–500 cc)  
 Large > 2 cm (> 500 cc)  
 Location: 4C ⇒ anterior RV/LV  
 Bicaval ⇒ atria  
 TG ⇒ posterior RV/LV  
 Chamber collapse RV, RA



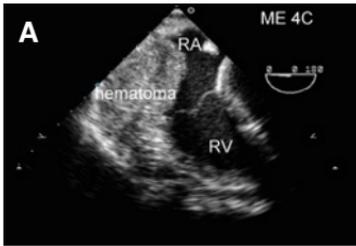
A large pericardial effusion is seen (A) posteriorly in the TG mid SAX and (B) laterally in the ME 4C view in a patient after a MVR. Compare TG SAX views showing (C) fibrinous pericardial effusion and (D) a patient with ascites (arrow) and a trace pericardial effusion.

## Cardiac Tamponade

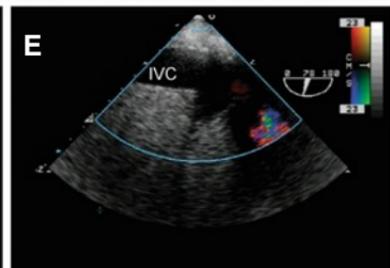
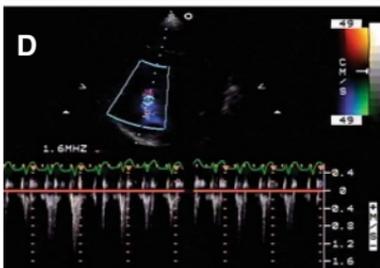
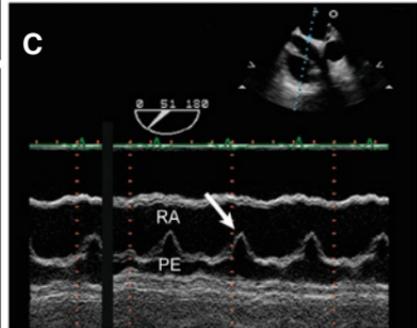
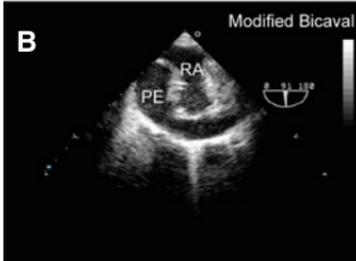
### Cardiac Tamponade

Tamponade is a **clinical** diagnosis. Clinical exam + TEE help exclude tamponade.

1. Pericardial effusion (moderate-large) with hemodynamic consequences
2. Chamber collapse occurs when intracavitary pressures are lowest
  - RA systolic collapse ( $> 1/3$  systole sensitive and specific)
  - RV diastolic collapse, less specific but more sensitive
  - Rarely see LA or LV collapse
3. Respiratory variation in diastolic filling, for spontaneous respiration and opposite in ventilated patients.
  - RV inflow  $\uparrow$  insp. /  $\downarrow$  exp
  - LV inflow  $\downarrow$  insp. /  $\uparrow$  exp
  - Transvalvular flow changes: MV  $> 25\%$ , TV  $> 40\%$
4. IVC plethora (dilated),  $< 50\%$  inspiratory collapse with spontaneous ventilation



(A) ME 4C shows large extra-cardiac hematoma compressing the RA and TV. The hematoma is echodense, a common appearance after cardiac surgery. (B) Pericardial effusion (PE) surrounding the right atrium (RA) which collapses during systole as best shown with (C) M-mode (arrow).

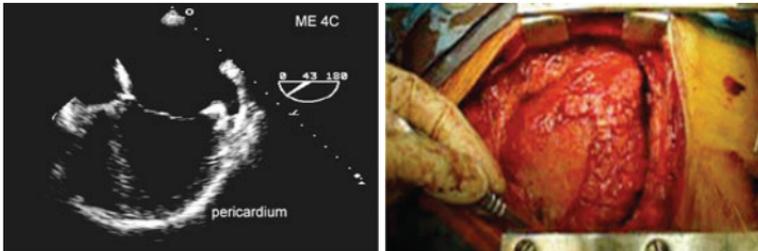


Additional findings include (D) 25% respiratory variation in TV inflow and (E) a dilated IVC without significant respiratory variation in a ventilated patient.

## Pericarditis

### Constrictive Pericarditis

- Etiology: idiopathic, radiation, post cardiac surgery, TB, renal failure, trauma
- Pericardial thickening > 4mm, visceral and parietal layers fused, no effusion
- Normal LV size and function
  - LA, RA can be normal or enlarged
- Dilated IVC, no inspiratory collapse with spontaneous respiration
- Difficult TG views due to pericardial calcification
- Doppler: Color: TR, diastolic MR
  - Spectral: LV inflow E>>A (diastolic dysfunction)
  - Respiratory variation: 25% MV and TV inflows
- Pulmonary veins: systolic > diastolic flow with respiratory variation



ME 4C shows bright echogenic pericardium surrounding the LA and LV in a patient with constrictive pericarditis. Surgery involves a sternotomy without CPB. Cross-hatching of the visceral pericardium is performed on the LV before RV to minimize hemodynamic instability.

### Restrictive Cardiomyopathy

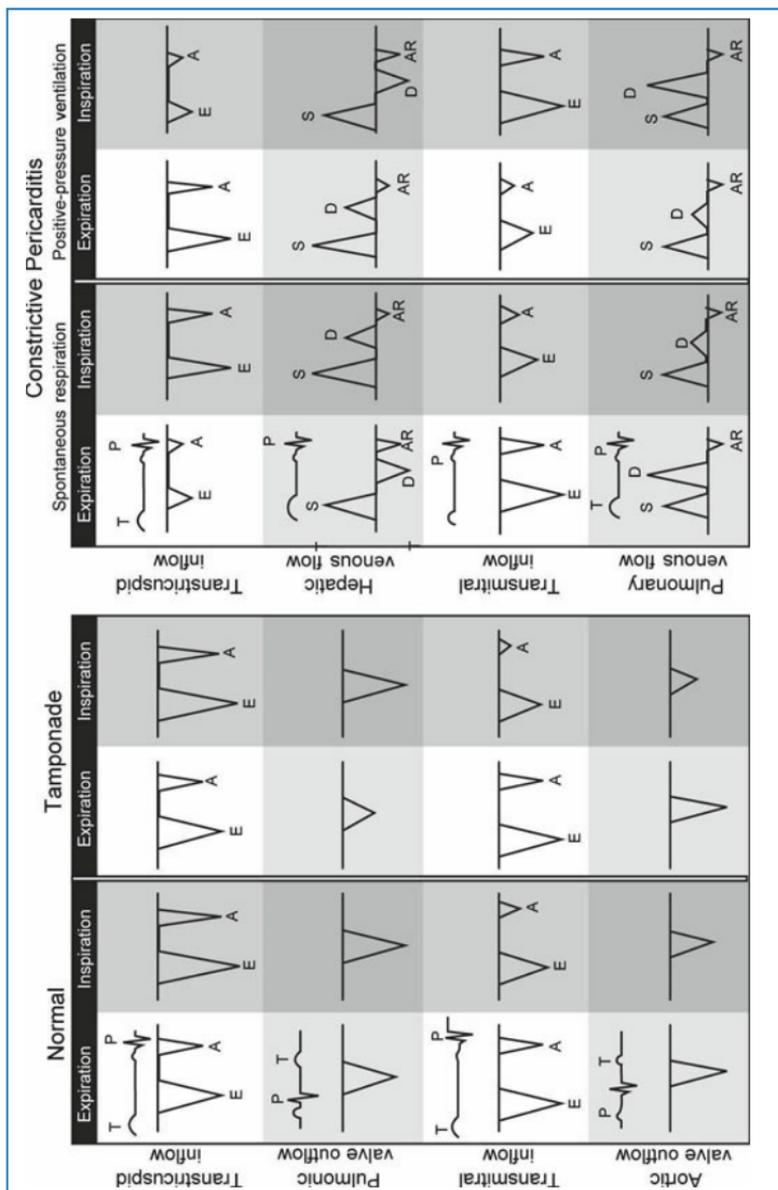
- Etiology: storage, infiltrative (sarcoidosis, amyloidosis), hypereosinophilic
- TEE Findings
  - Nondilated thickened LV, RV walls (concentric hypertrophy)
  - Biatrial enlargement
  - Normal systolic function
- Doppler: abnormal diastolic function (restrictive pattern)
  - moderate pulmonary hypertension (RVSP from TR)
  - lack of respiratory variation in MV inflow

### Pulsus Paradoxus

- > 10 mmHg variation in arterial pressures between inspiration and expiration
- Results from ↑ in venous return during inspiration → ↑ RV filling with shift of the IVS to the left and ↓ stroke volume
- Differential includes: tamponade, pulmonary embolism, cardiogenic shock, tension pneumothorax, SVC obstruction

	Tamponade	Constrictive	Restrictive
2D	Effusion	Thick pericardium	Thick V, dilated A
Respiratory variation	+	+	-
Diastolic dysfunction	No	Restrictive	Impair/restrict
IVC plethora	+	±	-

## Pericarditis



## Suggested Readings

### 1. Basic TEE Views

- Flachskampf FA, et al. Guideline from the Working Group: Recommendations for Performing Transesophageal Echocardiography. *Eur J Echocardiograph* 2001; 2:8-21.
- Shanewise JS, et al. ASE/SCA Guidelines for performing a comprehensive intraoperative multiplane transesophageal echocardiography examination. *Anesth Analg* 1999; 89:870-84.

### 2. Doppler and Hemodynamics

- Quinones MA, et al. Recommendations for quantification of Doppler echocardiography: a report from the Doppler Quantification Task Force of the Nomenclature and Standards Committee of the American Society of Echocardiography. *J Am Soc Echocardiogr* 2002; 15:167-84.

### 3. Ventricles

- Agricola E, et al. Ischemic mitral regurgitation: mechanisms and echocardiographic classification. *Eur J Echocardiogr* 2008;9:207-21.
- Cerqueira M, et al. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart: a statement for healthcare professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. *Circulation* 2002;105:539-42.
- Haddad F, et al. The right ventricle in cardiac surgery, a perioperative perspective: I. Anatomy, physiology, and assessment. *Anesth Analg* 2009;108:407-21.
- Lang RM, et al. Recommendations for chamber quantification: a report from the American Society of Echocardiography's Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. *J Am Soc Echocardiogr*. 2005; 18:1440-63.
- Rudski LG, et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. *J Am Soc Echocardiogr* 2010;7:685-713.
- Schiller NB, et al. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-Dimensional Echocardiograms. *J Am Soc Echocardiogr* 1989;2:358-87.

### 4. Native Valves

- Anyanwu A and Adams D. Etiologic classification of degenerative mitral valve disease: Barlow's disease and fibroelastic deficiency. *Semin Thorac Cardiovasc Surg* 2007;19: 90-96.
- Baumgartner H, et al. Echocardiographic assessment of valve stenosis: EAE/ASE recommendations for clinical practice. *J Am Soc Echocardiogr* 2009;22:1-23.
- Cohen GI, et al. Reference values for normal adult transesophageal echocardiographic measurements. *J Am Soc Echocardiogr* 1995;8:221-30.
- Eriksson MJ, et al. Mitral annular disjunction in advanced myxomatous mitral valve disease: echocardiographic detection and surgical correction. *J Am Soc Echocardiogr* 2005; 18:1014-22.
- Ho SY. Structure and anatomy of the aortic root. *Eur J Echocard* 2009;10:i3-10.
- Omran AS, et al. Intraoperative transesophageal echocardiography accurately predicts mitral valve anatomy and suitability for repair. *J Am Soc Echocardiogr* 2002; 15:950-7.
- Wilkins G. Percutaneous balloon dilatation of the mitral valve: an analysis of echocardiographic variables related to outcome and the mechanism of dilatation. *Br Heart J* 1988; 60:299-308.
- Zoghbi W, et al. Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. *J Am Soc Echocardiogr* 2003;16:777-802.

### 5. Prosthetic Valves, Transcatheter Valves, and Valve Repairs

- Cohen GI et al. Color Doppler and two-dimensional echocardiographic determination of the mechanism of aortic regurgitation with surgical correlation. *J Am Soc Echocardiogr* 1996;9:508-15.
- El Khoury G, et al. Functional classification of aortic root/valve abnormalities and their correlation with etiologies and surgical procedures. *Curr Opin Cardiol* 2005; 20:115-21.

## Suggested Readings

- Foster GP, et al. Accurate localization of mitral regurgitant defects using multiplane transeophageal echocardiography. *Ann Thoracic Surg* 1998; 65:1025-31.
  - Moss RR, et al. Role of echocardiography in percutaneous aortic valve implantation. *JACC Cardiovasc Imaging* 2008;1:15-24.
  - Zoghbi W, et al. Recommendations for evaluation of prosthetic valves with echocardiography and Doppler ultrasound: a report From the ASE Guidelines and Standards Committee and the Task Force on Prosthetic Valves, developed in conjunction with the ACC Cardiovascular Imaging Committee, Cardiac Imaging Committee of the AHA, the European Association of Echocardiography, a registered branch of the ESC, the Japanese Society of Echocardiography and the Canadian Society of Echocardiography, endorsed by the ACC Foundation, AHA, European Association of Echocardiography, a registered branch of the ESC, the Japanese Society of Echocardiography, and Canadian Society of Echocardiography. *J Am Soc Echocardiogr* ;2009; 22:975-1014.
- 6. Aorta**
- Evangelista A, et al. Echocardiography in aortic diseases: EAE recommendations for clinical practice. *Eur J Echocardiogr*. 2010;11(8):645-58.
  - Glas K, et al. Guidelines for the performance of a comprehensive intraoperative epiaortic ultrasonographic examination: recommendations of the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists; endorsed by the Society of Thoracic Surgeons. *J Am Soc Echocardiogr* 2007;11:1227-35.
  - Orihashi K, et al. Aortic arch branches are no longer a blind zone for transesophageal echocardiography: a new eye for aortic surgeons. *J Thor Card Surg* 2000;120:460-72.
- 7. Congenital Heart Disease**
- Russell IA, et al. Congenital heart disease in the adult: a review with internet-accessible transeophageal echocardiographic images. *Anesth Analg* 2006; 102: 694-723.
  - Shina A, et al. Two-dimensional echocardiographic–surgical correlation in Ebstein's anomaly: preoperative determination of patients requiring tricuspid valve plication vs replacement. *Circulation* 1983; 68:534-44.
- 8. Variants, Foreign Material, Masses, and Endocarditis**
- Baddour L, et al. Infective endocarditis: diagnosis, antimicrobial therapy, and management of complications: a statement for healthcare professionals from the Committee on Rheumatic Fever, Endocarditis, and Kawasaki Disease, Council on Cardiovascular Disease in the Young, and the Councils on Clinical Cardiology, Stroke, and Cardiovascular Surgery and Anesthesia, American Heart Association: endorsed by the Infectious Diseases Society of America. *Circulation* 2005;111:e394-e434.
  - Durack DT, et al. New criteria for diagnosis of infective endocarditis: utilization of specific echocardiographic findings. Duke Endocarditis Service. *Am J Med* 1994;96:200-9.
  - Goyal SK, et al. Persistent left superior vena cava: a case report and review of literature. *Cardiovasc Ultrasound* 2008; 6:50.
  - Tazelaar HD, et al. Pathology of surgically excised primary cardiac tumors. *Mayo Clin Proceed* 1992;67:957-65.
  - Wilson W, et al. Prevention of infective endocarditis: guidelines from the American Heart Association: a guideline from the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. *Circulation* 2007; 116:1736-54.
- 9. Ventricular Assist Devices and Heart Transplantation**
- Chumnanvej S, et al. Perioperative echocardiographic examination for ventricular assist device implantation. *Anesth Analg* 2007;106:583-401.
- 10. Cardiomyopathy and Diastolic Function**
- Nagueh SF, et al. Recommendations for the evaluation of left ventricular diastolic function by echocardiography. *J Am Soc Echocardiogr* 2009;2:107-33.
  - Skubas N. Intraoperative Doppler tissue imaging is a valuable addition to cardiac anesthesiologists' armamentarium: a core review. *Anesth Analg* 2009;108:48-66.

# Index

## A

- Abscess, 194
- Acyanotic heart disease, 154
- Adventitia, 140
- Alfieri repair, 133
- Aliasing
  - color Doppler, 33
  - spectral Doppler, 35
- Anastomosis
  - heart failure, 206, 207
  - pulmonary vein, 42
- Aneurysm
  - aortic, 143
  - aortic root, 145
  - ascending aortic, 145
  - ventricle, 72
- Annular dilatation, 135
- Annuloaortic ectasia, 145
- Annuloplasty, 132, 135
- Anterior leaflet repair, 133
- Aorta
  - anatomy, 138
  - aortic arch arteries, 139
  - ascending, 24, 25
  - descending, 20, 21
  - TEE views, 138
  - valve sparing procedure, 144–145
- Aortic aneurysm, 143
- Aortic arch, 22, 23, 139
- Aortic cannula, 203
- Aortic dissection, 148–149
- Aortic insufficiency (AI)
  - clinical findings, 86
  - color Doppler, 89
  - cuspid prolapse, 87
  - etiology, 86
  - jets, 87–89
  - mitral valve, indirect effects on, 87
  - severity, 86
  - spectral Doppler tracings, 89
  - VAD, 201
- Aortic root aneurysm, 145
- Aortic stenosis (AS)
  - calcified vs. rheumatic aortic valve, 83
  - etiology, 82
  - image findings, 82–83
  - indications for surgery, 85
  - LVOT, 84–85
  - planimetry, 85
  - prognosis, 85
  - stenosis severity, 82
  - AS velocity, 84
- Aortic valve, 10, 11. *See also* Aortic insufficiency (AI); Aortic stenosis (AS)
  - aortic root, 78
  - bicuspid, 81
  - normal aortic valve, 78
  - prosthesis, 124
  - repairs, 134–135
  - TEE views, 79–80
- Artificial chordae, 132
- Ascending aortic aneurysm, 145
- Atheroma, 140
- Atrial septal defects
  - cleft mitral valve, 158
  - 2D and Doppler imaging, 156
  - device closure, 157
  - primum, 158
  - secundum, 157
  - sinus venosus, 159
  - types, 156
- AV groove separation, 197
- Axial flow, 200

## B

- Baffle obstructions, 168–169
- Ball cage valve, 119
- Barlow's disease, 102
- Bi-atrial heart transplant, 206
- Bicaval heart transplant, 206
- Bicuspid aortic valve (BAV), 81
- Bileaflet valve, 119, 120
- Billowing leaflet, 98
- Bioprosthetic heart valves, 122–123

**C**

- Calcific valves, 83
- Cannula thrombus, 204
- Cannulation, 183
- Cardiac structures
  - aortic arch, 22, 23
  - aortic valve, 10, 11
  - ascending aorta, 24, 25
  - coronary sinus, 29
  - descending aorta, 20, 21
  - five chamber, 26
  - four chamber, 6
  - inferior vena cava, 27
  - left atrial appendage, 28
  - right ventricular inflow, 19
  - right ventricular outflow, 12
  - two chamber, 8, 16
- Cardiac tamponade, 204, 223
- Cardiopulmonary pressure, 44
- Catheter, 184
- Chronic segmental dysfunction, 71
- Coanda effect, 99
- Coarctation stent, 184
- Color Doppler
  - aliasing/flow acceleration, 33
  - assessment, 32
  - color scale, 33
  - Doppler artifacts, 36
  - Doppler indications, 35
  - normal, 32
  - parameters, 32
  - pulsed Doppler, 32
  - turbulent flow, 33
- Commissural prolapse and resuspension, 135
- Congenital heart disease
  - atrial septal defects (*see* Atrial septal defects)
  - classification, 154
  - cor triatriatum, 175
  - D-TGA, 166–167
  - Ebstein's anomaly, 172–173
  - Fontan procedure, 170–171
  - inter-atrial septum, 155
  - L-TGA, 174
  - mustard procedure, 168–169

- patent ductus arteriosum, 176
- patent foramen ovale, 155
- subaortic membran, 177
- TEE segmental approach, 152–153
- tetralogy of fallot (TOF), 164–165
- ventricular septal defect (*see* Ventricular septal defect)

- Constrictive pericarditis, 224–225
- Continuity equation, 45
- Coronary anatomy, 70
- Coronary sinus, 29
- Cor triatriatum, 175
- Cusp perforation, 135
- Cusp prolapse and resuspension, 135
- Cyanotic heart disease, 154

**D**

- Dacron graft, 144
- Dehiscence, 196
- Device deairing, 202
- Diastasis, 214
- Diastolic function
  - clinical manifestation, 215
  - color M mode transmitral flow, 217
  - definition, 215
  - diastolic phases, 214
  - 2D mode, 215
  - IVRT, 216
  - pulmonary vein flow Doppler, 216
  - tissue Doppler imaging, 217
  - transmitral Doppler flow, 216
- Doppler artifacts, 36. *See also* Color Doppler; Spectral Doppler
- Doppler effect, 34
- D-transposition of the great arteries (D-TGA), 166–167
- Duke criteria, 192

**E**

- Ebstein's anomaly, 172–173
- Effective regurgitant orifice area, 47
- Ejection fraction, 65
- Elephant trunk, 184
- El Khoury classification, 134
- Endocarditis
  - abscess, 194

- AV groove separation, 197
- complications, 192
- Duke criteria, 192
- fistula, 196, 197
- infective endocarditis, 192
- jet lesion, 196
- predisposition, 192
- prosthetic valve dehiscence, 196
- pseudoaneurysm, 195, 197
- vegetations, 193
- Epiaortic scanning, 141
- F**
- Fibroelastic degeneration, 103
- Fibroelastoma, 187
- Fistula, 196, 197
- Flail leaflet, 98
- Fontan procedure, 170–171
- Foreign material
  - cannulation, 183
  - coarctation stent, 184
  - elephant trunk, 184
  - intra-aortic balloon pump catheter, 184
  - pleural effusions, 183
  - pulmonary artery band, 184
- Fractional area change (FAC), 64
- Fractional shortening (FS), 64
- G**
- Gerbode defect, 163
- Global systolic function, 64
- H**
- Heart transplantation, 206–207
- Hematoma, intramural, 142
- Hypertrophic obstructive cardiomyopathy (HOCM)
  - asymmetrical vs. symmetrical hypertrophy, 210
  - clinical manifestation, 210
  - diastolic function (*see* Diastolic function)
  - pathophysiology, 210–211
  - post-myectomy, 213
  - TEE findings, 212–213
- I**
- Imaging planes, 4
- Impaired relaxation, 215
- Infective endocarditis, 192
- Inferior vena cava, 27
- Inflow cannula obstruction, 204
- Inter-atrial septum and PFO, 155
- Intima, 140
- Intracavitary thrombus, 201
- Intramural hematoma, 142
- Intraventricular pressure (dP/dt), 64
- Ischemic complications
  - aneurysm, 72
  - chronic segmental dysfunction, 71
  - ischemic mitral valve, 75
  - LV ventricular dilatation, 71
  - mitral regurgitation, 74
  - papillary muscle rupture, 74–75
  - pseudoaneurysm, 72
  - thrombus, 71
  - ventricular septal defect, 73
- Isovolumic relaxation time (IVRT), 214, 216
- J**
- Jet lesion, 196
- L**
- Left atrial appendage, 28
- Left superior vena cava (LSVC), 182
- Left ventricle
  - AHA 17 segment LV model, 54–55
  - SCA/ASE 16 segment LV model, 52–53
- Left ventricular assist device (LVAD), 201
- Left ventricular diastolic function, 214
- L-transposition of the great arteries (L-TGA), 174
- LV apical cannula, 203
- LV decompression, 202
- LV ventricular dilatation, 71
- M**
- Masses
  - thrombi (*see* Thrombus)

- tumor (*see* Tumor)
- Mechanical heart valves
  - aortic position, 121
  - ball cage, 119
  - bileaflet, 119, 120
  - mitral position, 120
  - tilting disc, 119, 121
  - valved conduit, 119
- Media, 140
- Mitral commissural, 7
- Mitral regurgitation (MR), 74
  - anterior mitral regurgitation jet, 101–102
  - Barlow's mitral valve, 102–103
  - clinical findings, 96
  - CW Doppler, 97
  - etiology, 96
  - excessive mitral leaflet motion, 98
  - fibroelastic disease, 103
  - jet area mapping, 97
  - mitral valve prolapse, 98–100
  - PISA (EROA), 97
  - posterior mitral regurgitation jet, 100–101
  - severity assessment, 96–97
  - vena contracta, 97
- Mitral stenosis (MS)
  - clinical findings, 104
  - etiology, 104
  - restricted mitral valve, 106–107
  - severity assessment, 104–105
- Mitral valve. *See also* Mitral regurgitation (MR); Mitral stenosis (MS)
  - anatomy, 90–92
  - function, 92
  - orientation, 91
  - prosthesis, 125
  - repairs, 132–133
  - TEE views, 91, 93–95
- Mitral valve inflow, 216
- Mustard procedure, 168–169
- Myectomy, 213
- Myocardial ischemia, 71
- Myocardial performance index (RVMPI), 69
- Myxoma, 186
- N
- Native cardiac valve anatomy
  - aortic valve, 78
  - mitral valve, 90–92
  - pulmonic valve, 113
  - tricuspid valve, 108
- Normal variants
  - aortic and mitral valve, 180
  - artifacts, 181
  - bicaval view, 181
  - intra-atrial septum, 180
  - LAA view, 181
  - left and right atrium, 180
  - ME 2C, 181
  - ME 4C view, 181
  - pericardium, 180
  - persistent LSVC, 182
  - right and left ventricle, 180
- Nyquist limit, 33
- O
- Outflow cannula, 200, 204
- P
- Pacer thrombus, 190
- Papillary muscle rupture, 74–75
- Paravalvular leaks, 126
- Patch closure, 135
- Patent ductus arteriosus, 176
- Patent foramen ovale (PFO), 155, 201
- Patient prosthetic mismatch (PPM), 123
- Percutaneous ventricular assist devices, 205
- Pericardial effusion, 222
- Pericardial spaces, 220
- Pericarditis, 224–225
- Pericardium
  - anatomy, 220
  - cardiac tamponade, 223
  - effusion, 222
  - pericarditis, 224–225
  - TEE views, 221
- Pleural effusions, 183

- Plication, 135
  - Pressure gradients, 34, 38, 44, 45
  - Pressure half-time, 45–46
  - Prolapsed leaflet, 98
  - Prosthetic cardiac valves
    - bioprosthetic heart valves (*see* Bioprosthetic heart valves)
    - mechanical heart valves (*see* Mechanical heart valves)
    - normal prosthetic valve findings, 118
    - patient prosthetic mismatch, 123
    - pressure gradients, 118
    - types, 118
    - valve function (*see* function)
  - Prosthetic valve function
    - aortic valve prosthesis, 124
    - mitral and aortic positions, 126
    - mitral valve prosthesis, 125
    - paravalvular leaks, 126
    - prosthetic valve dysfunction, 127
    - pulmonic valve prosthesis, 125
    - tricuspid valve prosthesis, 125
    - washing (regurgitant) jets, 126
  - Proximal isovelocity area (PISA)
    - method, 46
  - Pseudoaneurysm, 72, 195–197
  - Pseudonormal diastolic dysfunction, 215
  - Pseudonormal pattern, 215
  - Pulmonary artery band, 184
  - Pulmonary emboli, 191
  - Pulmonary thomboendarterectomy, 191
  - Pulmonary vein flow, 216
  - Pulmonary veins, 42–43
  - Pulmonary venous baffle, 168–169
  - Pulmonic valve
    - anatomy, 113
    - prosthesis, 125
    - pulmonic insufficiency, 114
    - pulmonic stenosis, 115
  - Pulsatile flow, 200
  - Pulsus paradoxus, 224
- Q**
- Quadrangular resection, 133
- R**
- Regurgitant fraction, 48
  - Regurgitant volume, 48
  - Restrictive cardiomyopathy, 224
  - Restrictive diastolic dysfunction, 215
  - Rheumatic valves, 83
  - Right ventricle, 68–69
    - inflow, 19
    - outflow, 12
  - Right ventricular assist device (RVAD), 205
  - Right ventricular ejection fraction (RVEF), 69
  - Right ventricular fractional area change (RVFAC), 69
- S**
- Sectors, 4, 13, 16, 18
  - Segmental wall motion, 62
  - Septal hypertrophy, 210
  - Shunt fraction, 49
  - Sinus of Valsalva aneurysm (SOVA), 146–147
  - Speckle tracking, 67
  - Spectral Doppler. *See also* Spectral profiles
    - aliasing, 35
    - Doppler artifacts, 36
    - Doppler indications, 35
    - fast Fourier transformation, 34
    - parameters, 34
    - pulsed *vs.* continuous wave, 35
    - spectral broadening, 36
    - velocity measurement, 35
  - Spectral profiles
    - aortic insufficiency, 39
    - aortic stenosis, 39
    - aortic valve, 37
    - ascending aorta, 41
    - coronary artery, 40
    - coronary sinus, 40
    - descending aorta, 41
    - distal aortic arch, 41

- hepatic vein, 41
  - left atrial appendage, 40
  - mitral regurgitation, 38
  - mitral stenosis, 38
  - mitral valve, 37
  - pulmonary artery, 40
  - pulmonic insufficiency, 39
  - pulmonic stenosis, 39
  - pulmonic valve, 37
  - tricuspid regurgitation, 38
  - tricuspid stenosis, 38
  - tricuspid valve, 37
  - Standard TEE views, 2–3, 5
  - Stented bioprosthetic valve, 122
  - Stentless prosthetic valve, 123
  - Strain, 66
  - Stroke volume, 63
  - Subaortic membrane, 177
  - Systemic venous baffle, 168–169
  - Systolic anterior motion (SAM), 211
  - Systolic flow reversal, 42
- T**
- TEE. *See* Transesophageal echocardiography (TEE)
  - Tetralogy of Fallot (TOF), 164–165
  - Thrombus, 71
    - cannula, 204
    - intracavitary, 201
    - LAA thrombus, 190
    - pacer, 190
    - pacer thrombus, 190
    - pulmonary emboli, 191
    - pulmonary thromboendarterectomy, 191
    - transit, 190
  - Tilting disc valve, 119, 121
  - TOF. *See* Tetralogy of fallot (TOF)
  - Transcatheter aortic valve implantation (TAVI), 129
  - Transcatheter valves, 128–131
  - Transesophageal echocardiography (TEE)
    - mid-esophageal aortic valve long-axis, 10
    - mid-esophageal aortic valve short axis, 11
    - mid-esophageal ascending aorta long-axis, 25
    - mid-esophageal ascending aorta short-axis, 24
    - mid-esophageal bicaval, 13
    - mid-esophageal coronary sinus, 29
    - mid-esophageal descending aorta long-axis, 21
    - mid-esophageal descending aorta short-axis, 20
    - mid-esophageal five chamber, 26
    - mid-esophageal four chamber, 6
    - mid-esophageal left atrial appendage, 28
    - mid-esophageal long-axis, 9
    - mid-esophageal mitral commissural, 7
    - mid-esophageal right ventricular outflow, 12
    - mid-esophageal two chamber, 8
    - planes and display, 4
    - standard TEE views, 2–3, 5
    - transgastric basal short-axis, 14
    - transgastric deep long-axis, 18
    - transgastric inferior vena cava, 27
    - transgastric long-axis, 17
    - transgastric mid short-axis, 15
    - transgastric right ventricular inflow, 19
    - transgastric two chamber, 16
    - upper-esophageal aortic arch long-axis, 22
    - upper-esophageal aortic arch short-axis, 23
  - Transposition of the great arteries, 166–167, 174
  - Tricuspid annular plane systolic excursion (TAPSE), 69
  - Tricuspid valve
    - anatomy, 108–109
    - prosthesis, 125
    - tricuspid regurgitation, 110–111
    - tricuspid stenosis, 112
  - Tumor
    - diagnosis, 185

- etiology, 185
  - fibroelastoma, 187
  - myxoma, 186
  - renal cell tumor, 189
  - sarcoma, 188, 189
  - surgery, 185
- V**
- VAD. *See* Ventricular assist devices (VAD)
  - Valve area
    - aortic, 45
    - mitral, 46
  - Valved conduit, 119
  - Valve regurgitation
    - aortic insufficiency, 86–89
    - mitral regurgitation, 96–103
    - tricuspid regurgitation, 110–111
  - Valve repairs
    - aortic, 134–135
    - mitral, 132–133
  - Valve sparing procedure, 144–145
  - Valve stenosis
    - aortic, 82–85
    - mitral, 104–107
    - pulmonic, 115
    - tricuspid, 112
  - Vegetations, 193
  - Ventricular assist devices (VAD)
    - aortic insufficiency, 201
    - complication, 204
    - continuous flow VADs, 200
    - function, 202–203
    - heart transplantation, 206–207
    - intracavitary thrombus, 201
    - patent foramen ovale, 201
    - percutaneous VAD, 205
    - pulsatile VADs, 200
    - right ventricular assist device, 205
  - Ventricular dimensions, 56–61
  - Ventricular septal defect (VSD), 73
    - 2D and Doppler imaging, 160
    - Doppler flow, 163
    - Gerbode defect, 163
    - perimembranous inlet/outlet, 162
    - TEE views, 161
    - types, 160