SYLLABUS

11th Annual Comprehensive Review & Update of Perioperative Echo

ADVANCED

FEBRUARY 14-16, 2008

Sheraton San Diego Hotel & Marina

San Diego, CA

Society of Cardiovascular Anesthesiologists

In Cooperation With

American Society of Echocardiography

For cardiologists, cardiothoracic surgeons and cardiovascular anesthesiologists

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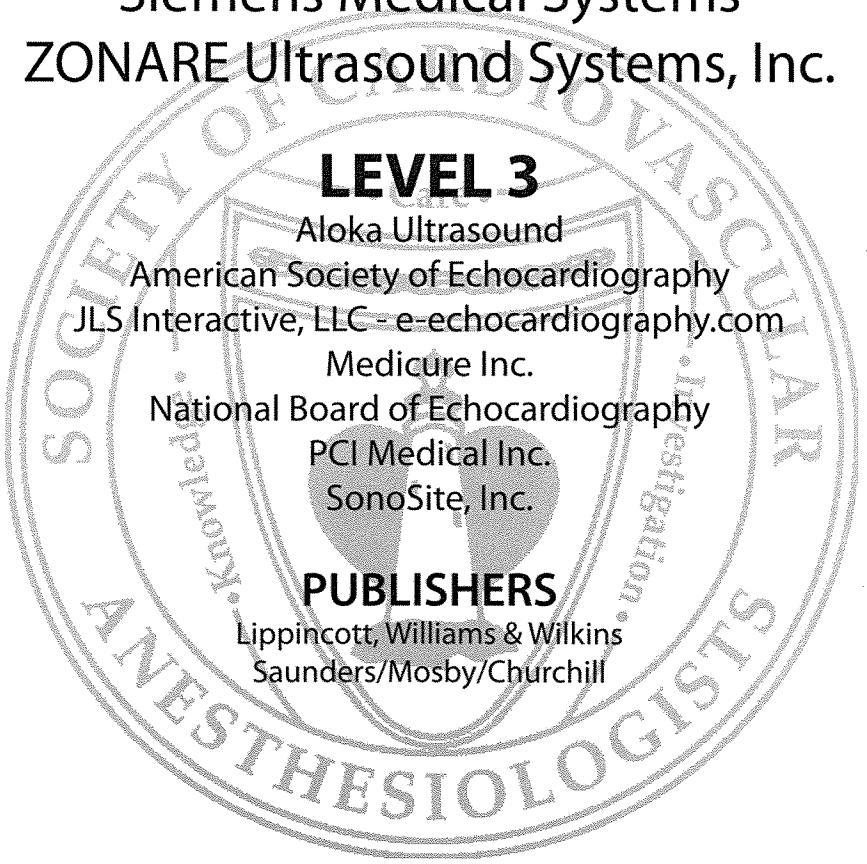
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Annual Comprehensive Update & Review of Perioperative Echo - ADVANCED

Clinical Decision Making in the Cardiac Surgery Patient

February 14-16, 2008 • San Diego, CA

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The Society of Cardiovascular Anesthesiologists designates this educational activity for a maximum of 25 ***AMA PRA Category 1 Credit(s)™*** . Physicians should only claim credit commensurate with the extent of their participation in the activity.

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SYMPOSIUM OVERVIEW

Welcome to the 11th Annual Comprehensive Review & Update of Perioperative Echocardiography: Clinical Decision Making in the Cardiac Surgery Patient, jointly sponsored by the Society of Cardiovascular Anesthesiologists and the American Society of Echocardiography. Over the past 11 years, this meeting has grown in size and caliber, and is now recognized as one of the premiere courses devoted to perioperative echocardiography. The historical success of this program is a result of its comprehensive structure which includes a review of the fundamentals of perioperative echocardiography and associated clinical applications, while highlighting novel, cutting-edge ultrasound technology, including the latest breakthroughs in transesophageal echocardiography, transthoracic echocardiography, epicardial and epiaortic imaging.

As Co-Chairmen of this educational collaboration, it is our great pleasure to continue the legacy of this meeting. As in the past, it has been our intention to continually modify and refine the course and its content. Many of these changes are the direct result of feedback from prior course participants. For those first-time attendees, we hope the meeting provides both a strong review of fundamental concepts of echocardiography as well as an introduction to new applications and frontiers of the modality and specialty. Returning participants will find changes to the basic format designed in an effort to streamline the workshops, refresh the presentations, and continue to provide the most comprehensive review and update on echocardiography. We recognize that it is important for all participants in this meeting to have an appropriate foundation and understanding of the basics of echocardiography in order to benefit from the more advanced applications of echocardiography in the perioperative clinical setting. Thus, this symposium is intentionally structured in a stepwise progression, beginning with a foundation of basic echocardiographic principles that will provide the novice echocardiographer with an introduction to transesophageal echocardiography through the Basic TEE & Surgical Anatomy Wet Lab courses. Workshops include an Anatomy Imaging Plane Workshop, Live Demonstration of Imaging Principles, Obtaining Basic Measurements and Values, Regional Wall Motion Analysis, and the Cardiac Anatomy Wet Lab. The Hemodynamics Workshops have been divided into basic and advanced and will help to consolidate fundamental concepts as well as address more advanced applications of quantitative echocardiography. The format of the anatomy workshop has been modified with the presentations being "structure" focused. Similar to last year, an intermission during lunch has been integrated. In addition, each session has been lengthened to provide an opportunity for a more coherent delivery of materials and permit a greater depth of presentation material.

Tuesday evening has been redesigned and focuses on the non-cardiac applications of ultrasound for clinical practice; specifically ultrasound guided regional anesthesia and vascular access. In addition to didactic sessions on each, the breakout workshops will include hands-on stations with "ultrasoundable" simulators utilized designed for individual practice and skill refinement. On Wednesday evening we will once again have an industry sponsored symposium, wherein this year, the leaders in industry will highlight the future of their respective technologies. In addition, the course directors and faculty will be available for discussion and comment for this session.

The remainder of the week focuses on more advanced principles directed towards the echocardiographic evaluation of valves, ventricular function and the role of echocardiography in facilitating perioperative decision-making. In addition, in recognition of the rapid progression of New Echocardiography Technology, sessions devoted towards three-dimensional echocardiography, Doppler tissue imaging and strain, and portable echocardiographic systems are once again be included in this year's meeting.

The format of the several sections of the advanced session has been changed. On Thursday afternoon, a one-hour session will be devoted to advanced clinical decision making with respect to the aortic valve. Clinical dilemmas such as moderate aortic stenosis for a patient with coronary artery disease, incidental aortic root dilation, etc. will be discussed by panelists from the three disciplines of cardiology, surgery and anesthesiology, each providing their unique input, concerns and factors integral to their decision making processes. A similar format will be utilized on Friday with cases involving the mitral valve. In addition to these lectures, presentations focused on echocardiography for heart failure, congenital heart disease and cardiomyopathies are incorporated into the advanced session. The mock echocardiography examination is once again on the final afternoon and night to serve as a "wrap-up" assessment opportunity. The session has been modified and will include a written question book. Finally, we recognize the importance of maintaining an interactive format between faculty and participants which will be encouraged through the integration of several Question/Answer and Case Discussion sessions throughout the week. Saturday's session is an opportunity for participants to serve as perioperative echocardiographic consultants by reviewing "unknown" case studies in an interactive format.

Two more important additions should be highlighted. Beginning both the basic and advanced sessions, cases will be presented as examples of the knowledge set for each section. At the conclusion of the basic and advanced courses, the final lecture will represent a detailed review of these same cases. This can serve as a "yard-stick" to measure your individual progression of knowledge and skills. Throughout the week during the coffee breaks, at the industry display areas, a booth will be staffed by faculty and entitled "ask the experts." This will provide a formal, regular opportunity to ask for clarification, expand on knowledge sets and otherwise pick the brains of the presenting faculty.

It is important to recognize the world class, multidisciplinary faculty who will once again be available throughout the week to provide further comprehensive insight into the field of perioperative ultrasound. A key attraction of this year's meeting is the presentation of the first Annual Arthur Weyman Lecture sponsored by the National Board of Echocardiography in recognition of Dr. Weyman's outstanding contribution to the field of echocardiography. We are honored to have Dr. Weyman present to deliver his perspective on the Future of Echocardiography as the first lecturer in this annual series.

Finally, this year marks the last year in which we will be the course directors. As we begin the transition to pass the reins, we want to welcome the new Co-Chairs, Drs. Kathy Grichnik and Chris Troianos, and give them our best wishes. We also want to thank all the loyal faculty and participants who have significantly contributed to the success of this educational endeavor over the past years. We are confident that while this meeting will continue to evolve, its outstanding quality will remain consistent in the future.

We as course directors wish you all an exciting week of learning focused on the development of your echocardiographic skills.

Gregg S. Hartman, MD
Co-Director

Stanton K. Shernan, MD
Co-Director

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1. Nothing to disclose
2. Grants/research support
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4. Board membership
5. Consultant
6. Stock shareholder–directly purchased
7. Other financial/material support

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In compliance with the Accreditation Council for Continuing Medical Education (ACCME), all faculty members disclosing a relationship with industry for this meeting have supplied the Course Directors with a plan for managing their potential conflict. If you wish to see the management plans submitted, please feel free to contact the SCA's headquarters office.

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- Dynamic Medical Education 5
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11th Annual Comprehensive Review & Update of Perioperative Echo

Statement of Need

Ultrasonography has become an integral tool for the perioperative management of surgical patients. Echocardiography, in particular, remains the fundamental diagnostic modality and monitor of perioperative cardiac performance, however its utility is no longer limited to cardiac surgical patients. Non-cardiac surgical applications of perioperative echocardiography are rapidly becoming appreciated and incorporated into routine practice. Furthermore applications of perioperative ultrasound have extended beyond echocardiography to include imaging of non-cardiac vascular structures and the facilitation of peripheral nerve blockade. In order to utilize these modalities, comprehensive knowledge and expertise in the ultrasound technology and its clinical applications are essential to optimally guide surgical procedures, and direct medical management of patients with cardiovascular dysfunction. The members of the Society of Cardiovascular Anesthesiologists (SCA) and the American Society of Echocardiography (ASE) have mandated interdisciplinary, patient-oriented educational programs to teach the requisite skills necessary to use these diagnostic and monitoring tools effectively, and to assure a positive impact on patient care. This interdisciplinary program sponsored by the SCA and offered in cooperation with the ASE has been developed to meet the growing demand for such educational needs.

Target Audience

This course is designed for anesthesiologists, cardiologists, cardiac surgeons, and sonographers as a comprehensive review of perioperative echocardiography.

Advanced (February 14-16)

Course Objectives

The program will focus on the advanced principles and perioperative applications of ultrasound technology. The program will include an in-depth review of the influence of perioperative echocardiography on the anesthetic management and surgical decision-making process of patients undergoing cardiac and non-cardiac surgical procedures utilizing didactics and interactive case presentations.

Upon completion of this course, the participant will be capable of understanding the application of advanced echocardiographic diagnostic principles in the surgical patient including:

- ventricular function and regional wall motion assessment and its correlation with other diagnostic studies such as coronary angiography;
- the role of diastolic dysfunction in heart failure;
- the pathophysiology of valvular heart disease and perioperative assessment;
- newer echocardiographic techniques including three-dimensional echocardiography, stress, strain and strain rate, myocardial viability assessment;
- the role of perioperative echocardiography used in guiding surgical decision making for cardiac patients undergoing myocardial revascularization, valve repair or replacement, myomectomy for left ventricular outflow tract obstruction, congenital heart disease, thoracic aortic repair, biventricular resynchronization and procedures for heart failure.
- the echocardiographic evaluation of ventricular assist devices and the role of TEE in the separation from cardiopulmonary bypass;
- the interaction between the different specialties: surgeons, cardiologists and anesthesiologists in complex decision making processes involved in the perioperative care of surgical patients with cardiovascular disease.

Designation Statement

The Society of Cardiovascular Anesthesiologists designates this educational activity for a maximum of 25 **AMA PRA Category 1 Credit(s)™**. Physicians should only claim credit commensurate with the extent of their participation in the activity.

MEETING AT A GLANCE

Thursday, February 14, 2008

Advanced Echocardiography I

6:45 – 7:30 am	Continental Breakfast
7:30 – 7:40 am	**Announcements** GS Hartman/SK Shernan

Ventricular Function

Moderators: SK Shernan/C Troianos

7:40 – 8:30 am	**Advanced Challenging Cases** KP Grichnik, CA Troianos
8:30 – 9:00 am	**Advanced Systolic Function** AT Cheung
9:00 – 9:30 am	**Advanced Diastolic Function: "Beyond the E- and A-Wave"** M Swaminathan
9:30 – 10:00 am	**Tissue Doppler/Stress/Strain** NJ Skubas
10:00 – 10:30 am	Coffee Break with Exhibitors
10:30 – 11:00 am	**3D and 4D Echocardiography** D Shook
11:00 - 12:00 noon	**Case Discussions with Faculty** AT Cheung, M Swaminathan, NJ Skubas, D Shook, SK Shernan

Luncheon Series (box lunch served)

12:00 – 12:15 pm	Box lunches served
12:15 – 1:30 pm	**First Annual Arthur E. Weyman, MD Lecture** AE Weyman

Echocardiography for Heart Failure and Cardiomyopathies

Moderator: AD Maslow

1:30 – 2:00 pm	**Echocardiographic Evaluation of Cardiomyopathies** AD Maslow
2:00 – 2:30 pm	**Pericardial Disease** EG Avery
2:30 – 3:00 pm	**Intraoperative Echo for Ventricular Assist Devices and Heart Transplantation** TM Burch
3:00 – 3:30 pm	Coffee Break with Exhibitors

Advanced Echocardiography II: Aortic Valve – Advanced

Moderator: BA Bollen

3:30 – 4:00 pm	**Timing of Surgery/Intervention in Asymptomatic Aortic Valve Disease** AE Weyman
4:00 – 4:30 pm	**Patient Prosthetic Mismatch and Pressure Recovery after Aortic Valve Replacement** I Kronzon
4:30 – 5:00 pm	**Aortic Valve Replacement or Repair?** NG Smedira
5:00 – 6:00 pm	**Case Discussions with Faculty** BA Bollen, AE Weyman, I Kronzon, N Smedira
6:00 – 7:00 pm	Dinner on own

Advanced Hemodynamic Workshop

Moderators: AT Cheung/AD Maslow

7:00 – 7:30 pm	**Review of Advanced Hemodynamic Principles and Quantitative Doppler** AD Maslow
7:30 – 9:30 pm	**Advanced Hemodynamic Case Discussions** A Cheung, SK Shernan, SN Konstadt, CG Koch, AD Maslow

Friday, February 15, 2008

Advanced Echocardiography III

7:00 – 7:45 am	Continental Breakfast with Exhibitors
7:45 – 8:00 am	**Announcements** GS Hartman/SK Shernan

Advanced Mitral Valve

Moderator: BA Bollen

8:00 – 8:30 am	**Your Patient is Scheduled for Mitral Valve Repair but Presents with Only 2+ Regurgitation by Intraoperative TEE: What Now?** SN Konstadt
8:30 – 9:00 am	**Ischemic Mitral Regurgitation** S Aronson
9:00 – 9:30 am	**Surgical Repair of the Mitral Valve** Surgeon (TBA)
9:30 – 10:30 am	**Case Discussions with Faculty** BA Bollen, SN Konstadt, S Aronson, Surgeon TBA
10:30 – 11:00 am	Coffee Break with Exhibitors

Role of Echocardiography in Perioperative Clinical Decision-Making: Part I

Moderator: RM Savage

11:00 – 11:30 am	**Role of Echocardiography in Percutaneous Approaches to Valve Dysfunction** RM Savage
11:30 – 12:00 noon	**Mitral Valve Dysfunction after Mitral Valve Repair** SK Shernan

Luncheon Series (box lunch served)

12:00 – 12:15 pm	Box Lunches Served
12:15 – 1:00 pm	**The Future of Cardiac Surgery** D Adams

MORE >>>>>>>>>>

MEETING AT A GLANCE

Role of Echocardiography in Perioperative Clinical Decision-Making: Part II

Moderator: RM Savage

1:00 – 1:30 pm	**How to Use Echocardiography to Help Wean from Bypass** TM Burch
1:30 – 2:00 pm	**Unanticipated Ascending Aortic Dilatation: How Big is Too Big?** AT Cheung
2:00 – 2:30 pm	**Echocardiographic Assessment of Myocardial Viability** M Swaminathan
2:30 – 3:00 pm	**Echocardiography for Adult Congenital Heart Disease** IA Russell
3:00 – 3:30 pm	**Stump the Panel: Unusual Cases from the Faculty** RM Savage, TM Burch, AT Cheung, M Swaminathan, IA Russell
3:30 – 4:00 pm	Coffee Break

"Test Yourself"

Moderator: DA Zvara

4:00 – 5:30 pm	**"Test Yourself" Mock Test** **Faculty:** DA Zvara, BC Spence, TM Burch, EG Avery, CG Koch
5:30 – 7:30 pm	Dinner on Own
7:30 – 9:30 pm	**Mock Exam Review** **Faculty:** DA Zvara, BC Spence, TM Burch, EG Avery, CG Koch

Saturday, February 16, 2008

You're the Intraoperative Echocardiography Consultant

Moderators: GS Hartman/SK Shernan

6:45 – 7:30 am	Continental Breakfast
7:30 – 8:30 am	**Review of Advanced Cases** GS Hartman, SK Shernan
8:30 – 10:30 am	**You're the Intraoperative Echocardiography Consultant** **Faculty:** K Glas, AD Maslow, CG Koch, GS Hartman, SK Shernan

Advanced Echocardiography I

6:45 – 7:30 am Continental Breakfast
7:30 – 7:40 am **Announcements**
GS Hartman/SK Shernan

Ventricular Function

Moderators: SK Shernan/C Troianos

7:40 – 8:30 am **Advanced Challenging Cases**
KP Grichnik, CA Troianos
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M Swaminathan
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NJ Skubas
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D Shook
11:00 - 12:00 noon **Case Discussions with Faculty**
AT Cheung, M Swaminathan, NJ Skubas, D Shook, SK Shernan

Luncheon Series (box lunch served)

12:00 – 12:15 pm Box lunches served
12:15 – 1:30 pm **First Annual Arthur E. Weyman, MD Lecture**
AE Weyman

Echocardiography for Heart Failure and Cardiomyopathies

Moderator: AD Maslow

1:30 – 2:00 pm **Echocardiographic Evaluation of Cardiomyopathies**
AD Maslow
2:00 – 2:30 pm **Pericardial Disease**
EG Avery
2:30 – 3:00 pm **Intraoperative Echo for Ventricular Assist Devices and Heart Transplantation**
TM Burch
3:00 – 3:30 pm Coffee Break with Exhibitors

Advanced Echocardiography II: Aortic Valve – Advanced

Moderator: BA Bollen

3:30 – 4:00 pm **Timing of Surgery/Intervention in Asymptomatic Aortic Valve Disease**
AE Weyman
4:00 – 4:30 pm **Patient Prosthetic Mismatch and Pressure Recovery after Aortic Valve Replacement**
I Kronzon
4:30 – 5:00 pm **Aortic Valve Replacement or Repair?**
NG Smedira
5:00 – 6:00 pm **Case Discussions with Faculty**
BA Bollen, AE Weyman, I Kronzon, N Smedira
6:00 – 7:00 pm Dinner on own

Advanced Hemodynamic Workshop

Moderators: AT Cheung/AD Maslow

7:00 – 7:30 pm **Review of Advanced Hemodynamic Principles and Quantitative Doppler**
AD Maslow
7:30 – 9:30 pm **Advanced Hemodynamic Case Discussions**
A Cheung, SK Shernan, SN Konstadt, CG Koch, AD Maslow

NOTES

Advanced Challenging Cases

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For those of you only participating in the advanced session, for the first time this year, we are introducing a set of challenging cases to begin the advanced sessions of the comprehensive review. We ask that you review and become familiar with these cases during the course of each session. Clips of ultrasound images will be available for independent review during the days of the course. At the end of this advanced section, the final lecture will be reviews of these cases. The goal will be to "set-the stage" for what is to be covered during the advanced session and then to provide an opportunity for measuring personal knowledge progression from the first to the last day.

In conjunction with these sessions, a second new offering will be the "ask the experts" booth. Throughout the week, a special venue will be provided and staffed by faculty of the course, making these "experts" available for review of topics and providing participants a specific opportunity to ask and hopefully receive clarification on a subject of their choosing.

NOTES

Advanced Systolic Function

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Multiplane 2-dimensional TEE permits the entire left ventricle to be imaged throughout the cardiac cycle. Direct imaging of the left ventricle throughout the cardiac cycle can be used to provide information about left ventricular wall thickness, chamber size, and contractile performance. TEE measurements of global systolic function are useful for clinical decision making because they provide information pertaining to prognosis, the risk of cardiac operations, the severity of valvular heart disease, the extent of coronary artery disease, and the responses to vasoactive or other cardiovascular drug therapy. For example, in the American Heart Association guidelines for the management of patients with valvular heart disease, the decision for aortic valve replacement in patients with chronic aortic regurgitation without symptoms of heart failure is contingent on measures of left ventricular ejection fraction and left ventricular diameters (Figure 4, ACC/AHA 2006 Guidelines for the Management of Patients with Valvular Heart Disease).

GLOBAL LEFT VENTRICULAR SIZE AND SYSTOLIC FUNCTION

The normal left ventricle is a muscular organ of nearly uniform wall thickness surrounding a cavity with a circular cross section. The cross sectional area of the left ventricular cavity is greatest at the base and decreases toward the apex. Contraction of myocardial fibers during systole causes an increase in left ventricular wall thickness and a decrease in both the diameter and the length of the left ventricular chamber. The mitral valve leaflets, chordae tendineae, papillary muscles, and left ventricular apex provide anatomical landmarks for establishing the level of the cross-sectional imaging planes during the 2-dimensional echocardiographic examination. The entire left ventricle can be examined by TEE using the transgastric short-axis imaging planes at the level of the left ventricular apex, mid-papillary muscles, or left ventricular base. Alternatively, the entire left ventricle can also be examined using the TEE mid-esophageal long-axis imaging planes at multiplane angles of 0-20° (4-chamber), 80-100° (2-chamber), or 120-160° (long-axis).

Chronic systolic pressure overload caused by hypertension or aortic stenosis produces concentric left ventricular hypertrophy. In patients with compensated left ventricular hypertrophy, left ventricular mass increases relative to the cavity volume. This change can be most readily detected by TEE as an increase in left ventricular wall thickness. A left ventricular end-diastolic posterior wall thickness greater than 1.1 cm suggests left ventricular hypertrophy.

Chronic volume overload caused by congestive heart failure, aortic regurgitation, or mitral regurgitation also causes left ventricular hypertrophy. In contrast to chronic pressure overlad, volume overload results in an increase in left ventricular cavity size without an increase in ventricular wall thickness. An end-diastolic short-axis left ventricular cavity diameter greater than 5.5 cm or cross-sectional area greater than 22 cm^2 at the mid-papillary level suggests left ventricular dilation or eccentric hypertophy.

Left ventricular end diastolic and end systolic cavity volume can be measured from the mid-esophageal left ventricular 4 chamber and 2 chamber views using Simpson's rule. This measurement is accomplished by tracing the left ventricular endocardial border up to the mitral valve annulus and then using the calipers to measure the length of the left ventricular cavity from the mitral valve annulus to the apex. The accuracy of TEE for quantifying left ventricular volume is not as well established as with TTE. Foreshortening of the left ventricular cavity occurs if the long-axis cross sectional imaging plane is not directed through the true left ventricular apex and will cause underestimation of the actual left ventricular cavity volume. Representative normal echocardiographic values measured using Simpson's rule are: LV end-diastolic volume of 56-155 ml, LV end-systolic volume of 19-58 ml, and LV ejection fraction of $\geq$ 55%.

Reference values for left ventricular dimensions measured by echocardiography have recently been published in a consensus report from the American Society of Echocardiography. In that report, dimensions are indexed by gender, height, and body surface area. It is also important to note that the published reference dimensions were obtained from transthoracic echocardiographic studies measuring left ventricular dimensions at the mitral valve chordal level rather. TEE measurements often obtained at the transgastric left ventricular mid-papillary muscle level may yield slightly smaller dimensions.

Table 4 Reference limits and partition values of left ventricular mass and geometry

	Women				Men			
	Reference range	Mildly abnormal	Moderately abnormal	Severely abnormal	Reference range	Mildly abnormal	Moderately abnormal	Severely abnormal
Linear Method								
LV mass, g	67–162	163–186	187–210	≥211	88–224	225–258	259–292	≥293
LV mass/BSA, g/m²	*43–95*	*96–108*	*109–121*	*≥122*	*49–115*	*116–131*	*132–148*	*≥149*
LV mass/height, g/m	41–99	100–115	116–128	≥129	52–126	127–144	145–162	≥163
LV mass/height$^{2.7}$, g/m$^{2.7}$	18–44	45–51	52–58	≥59	20–48	49–55	56–63	≥64
Relative wall thickness, cm	0.22–0.42	0.43–0.47	0.48–0.52	≥0.53	0.24–0.42	0.43–0.46	0.47–0.51	≥0.52
Septal thickness, cm	*0.6–0.9*	*1.0–1.2*	*1.3–1.5*	*≥1.6*	*0.6–1.0*	*1.1–1.3*	*1.4–1.6*	*≥1.7*
Posterior wall thickness, cm	*0.6–0.9*	*1.0–1.2*	*1.3–1.5*	*≥1.6*	*0.6–1.0*	*1.1–1.3*	*1.4–1.6*	*≥1.7*
2D Method								
LV mass, g	66–150	151–171	172–182	≥193	96–200	201–227	228–254	≥255
LV mass/BSA, g/m²	*44–88*	*89–100*	*101–112*	*≥113*	*50–102*	*103–116*	*117–130*	*≥131*

BSA, Body surface area; LV, left ventricular; 2D, 2-dimensional.
Bold italic values: Recommended and best validated.

From: J Am Soc Echocardiogr 2005;18:1440-1463.

Table 5 Reference limits and partition values of left ventricular size

	Women				Men			
	Reference range	Mildly abnormal	Moderately abnormal	Severely abnormal	Reference range	Mildly abnormal	Moderately abnormal	Severely abnormal
LV dimension								
LV diastolic diameter	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
LV diastolic diameter/BSA, cm/m²	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
LV diastolic diameter/height, cm/m	2.5–3.2	3.3–3.4	3.5–3.6	≥3.7	2.1–3.3	3.4–3.5	3.6–3.7	≥3.8
LV volume								
LV diastolic volume, mL	56–104	105–117	118–130	≥131	67–155	156–178	179–201	≥201
LV diastolic volume/BSA, mL/m²	***35–75***	***76–86***	***87–96***	***≥97***	***35–75***	***76–86***	***87–96***	***≥97***
LV systolic volume, mL	19–49	50–59	60–69	≥70	22–58	59–70	71–82	≥83
LV systolic volume/BSA, mL/m²	***12–30***	***31–36***	***37–42***	***≥43***	***12–30***	***31–36***	***37–42***	***≥43***

BSA, body surface area; *LV*, left ventricular.
Bold italic values: Recommended and best validated.

Table 6 Reference limits and values and partition values of left ventricular function

	Women				Men			
	Reference range	Mildly abnormal	Moderately abnormal	Severely abnormal	Reference range	Mildly abnormal	Moderately abnormal	Severely abnormal
Linear method								
Endocardial fractional shortening, %	27–45	22–26	17–21	≤16	25–43	20–24	15–19	≤14
Midwall fractional shortening, %	15–23	13–14	11–12	≤10	14–22	12–13	10–11	≤10
2D Method								
Ejection fraction, %	***≥55***	***45–54***	***30–44***	***<30***	***≥55***	***45–54***	***30–44***	***<30***

2D, Two-dimensional.
Bold italic values: Recommended and best validated.

From: J Am Soc Echocardiogr 2005;18:1440-1463.

Global indexes of left ventricular systolic function can be measured from the TEE images based on the relative speed and change in end-diastolic and end-systolic left ventricular cavity size. The transgastric left ventricular short axis end diastolic area (EDA) and end systolic area (ESA) are are often used for this purpose and are measured by manual planimetry of the area bounded by the endocardium. The end systolic diameter (ESD) is approximately 2.8 cm and the ESA is approximately 6.2 cm^2 in normal adults.

1. Fractional shortening (FS) is the percent decrease in left ventricular cavity short axis diameter from end diastole (LVIDed) to end systole (LVIDes) and can be measured from the endocardial border or the midwall of the left ventricle.

$$FS = (LVIDed - LVIDes)/LVIDed \times 100 \quad \text{(Normal range = 14-45\%)}$$

2. Fractional area change (FAC) is the percent decrease in the left ventricular cavity cross-sectional area from end-diastole to end-systole.

$$FAC\ (\%) = \frac{(EDA \quad ESA)}{EDA} \quad 100 \qquad \text{(Normal = 57\%).}$$

3. Mean velocity of circumferential fiber shortening (Vcf)

$$Vcf\ (\text{circumference/second}) = \frac{EDD \quad ESD}{EDD \quad LVET} \quad 100$$

where EDD = end-diastolic LV cavity diameter,

ESD = end-systolic LV cavity diameter, and
LVET = left ventricular ejection time (seconds).

4. Heart rate corrected mean velocity of circumferential fiber shortening (Vcfc)

$$\text{Vcfc (circ/s)} = \frac{\text{Vcf}}{\sqrt{\text{R R interval}}} \quad \text{(Normal } \blacksquare \text{ 1.1 circ/s)}$$

All of these indexes of global ventricular function are based on a single cross-section of the left ventricle. The presence of ventricular asymmetry or dysynergy, especially when abnormal regions are not within the plane of the image used to calculate the index may render the index a poor indicator of global left ventricular function. Corrections to the global left ventricular ejection fraction can be estimated based on the presence of akinetic or dyskinetic regions outside of the imaging plane. For example, global left ventricular ejection fraction estimated from the transgastric mid-ventricular short axis image should be decreased by approximately 10% in the presence of an akinetic left ventricular apex.

Alternatively, a qualitative estimation of global ventricular function can be made based on visual inspection. Global ventricular function is graded as being normal, mildly, moderately, or severely impaired based on estimation of the left ventricular ejection fraction. The advantages of visual assessment are that it is based on multiple views, incorporates multiple parameters of left ventricular function, and can be performed immediately without the need for off-line image analysis. Studies have demonstrated that visual assessment of left ventricular function, despite its limitations, correlate well with quantitative measurements and is a useful parameter for predicting cardiac risk and morbidity. An echocardiographic left ventricular ejection fraction less than 30% is consistent with severe left ventricular dysfunction based on clinical trials involving patients with heart failure. Subjective estimation of global ventricular function or left ventricular ejection fraction is dependent on the experience of the echocardiographer and inter-observer variability may exist depending upon the parameters used to grade ventricular function. The accuracy and consistency of subjective estimation of global ventricular function can be improved by calibrating the estimation with established methods of quantification.

One difficulty encountered with the assessment of left ventricular function is that measures of systolic performance are dependent on the ventricular loading conditions at the time of the study. An abnormally high systolic blood pressure increases left ventricular afterload and decreases the FAC or Vcfc. A reduction in preload decreases both left ventricular end diastolic and end systolic cavity size. For these reasons, it is necessary to plot indexes such as the FAC or Vcfc against the systolic blood pressure or left ventricular wall stress to obtain an afterload-independent assessment of ventricular contractility. Left ventricular meridional wall stress (ESWS) that incorporates measures of peak systolic blood pressure (SBP), left ventricular wall thickness (H), and left ventricular end-systolic cavity diameter (ESD) can be estimated with the following formula :

$$\text{ESWS } (\text{x}10^3 \text{ dynes/cm}^2) = \frac{(0.334)(SBP)(ESD)}{(H)(1 \quad H / ESD)}$$

Estimating load-independent indexes of left ventricular contractile performance is tedious to perform clinically, but has been used in clinical studies to demonstrate the increase in left ventricular contractility in response to inotropic agents or the decrease in ventricular contractility in response to volatile anesthetic agents. Despite the recognition that performance measures such as ejection fraction are load-depend, echocardiographic measures of global left ventricular function has proven prognostic value in the clinical setting.

CONCLUSION

Echocardiographic measurements of left ventricular wall thickness, cavity size, and segmental wall motion obtained by TEE can be used to assess global and regional left ventricular function. The information provided by TEE is an important supplement to hemodynamic and electrocardiographic measurements for the diagnosis of left ventricular hypertrophy, cardiomyopathy, and the consequences of myocardial ischemia and infarction. It is important to recognize that left ventricular volumes cannot be directly measured from a single cross-sectional image. It is also important to recognize that left ventricular systolic function is dynamic and changes in response to anesthetic agents, vasoactive drug therapy, intravascular volume status, and arterial blood pressure.

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Advanced Diastolic Function: "Beyond the E- and A- wave"

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Introduction

Congestive heart failure (CHF) is a significant burden on healthcare resources among developed countries. Approximately one-half of CHF cases may have normal systolic function, depending on diagnostic definition, and represent diastolic heart failure.[1] Since some degree of systolic dysfunction can be identified at a molecular level in almost all cases of CHF, those with apparently preserved systolic function are termed 'heart failure with normal ejection fraction', or HFNEF.[2] However, a much larger proportion of patients with heart disease in the community have diastolic dysfunction as assessed by echocardiography, with or without signs of heart failure. Even when without failure symptoms, diastolic dysfunction is accompanied by high rates of all-cause mortality.

The principal abnormality in these cases is a defect in the left ventricular diastolic pressure-volume relationship. This abnormality leads to increased filling pressures for any given preload, with increases in pulmonary vascular pressures with consequent signs and symptoms of heart failure. The important distinguishing feature of diastolic heart failure is that it has a better long-term prognosis, with about half the mortality rate of systolic heart failure, and a distinctly different treatment rationale. Its recognition in cardiac surgical patients is therefore of paramount importance to intraoperative echocardiographers. The simple estimation of ejection fraction therefore is insufficient when assessing global myocardial function.

Diastole

Diastole has both, an active and passive component. Diastolic function depends on *active* myocardial relaxation, and *passive* elastic recoil. While active myocardial relaxation depends on the decrease in cytoplasmic calcium and its dissociation from troponin, passive elastic recoil depends on myocardial architecture (stiffness, compliance). There are several factors that can affect either active or passive diastolic properties and lead to diastolic dysfunction and CHF. The gold standard for measurement of diastolic function is the

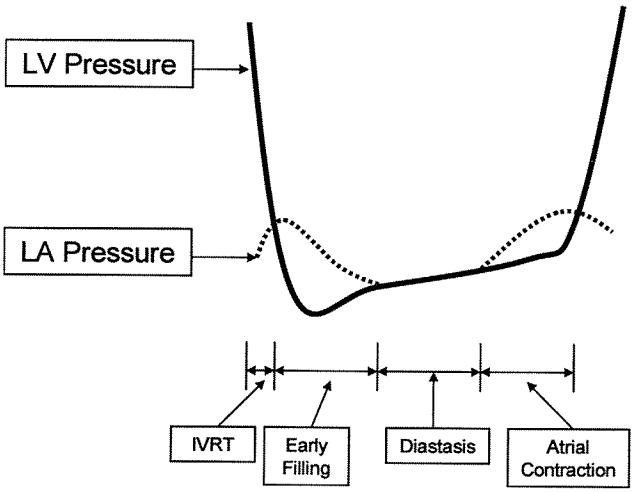


Figure 1: The four phases of diastole (see text for details)

examination of pressure-volume relationships over diastole. Since this is not feasible in daily clinical practice, echocardiography is the default standard for defining diastolic dysfunction. When it does occur, diastolic dysfunction can be recognized by the typical echocardiographic changes it produces. These will be presented in the following text.

IVRT	Early Filling	Diastasis	Atrial Contraction
• Continuum of the cardiac cycle • Systolic function • LV relaxation	• LV relaxation • Chamber compliance	• Heart rate • Chamber compliance	• Chamber compliance • LA function • Electrical conduction

Table 1: Factors affecting the four stages of diastole (IVRT = isovolumic relaxation time; LA = left atrium; LV = left ventricle)

Phases of Diastole

There are four distinct phases of diastole as and are described by changes in pressure and volume within the left ventricle (LV) (figure 1). Diastole begins with *isovolumic relaxation* and is continuous with the end of systole. The LV pressure is declining at this time and when it falls below the left atrial (LA) pressure, the mitral valve opens and the next phase of diastole – *early filling* begins. When the left atrial volume has been emptied into the left ventricle, the pressures in the two chambers equalize and no more flow occurs, marking the third phase of diastole – *diastasis*. This phase ends with *atrial contraction* – the fourth phase – when the left atrial pressure rises sharply, opening the mitral valve for the second time and filling the left ventricle. Diastole ends when the systolic phase begins with the rise in left ventricular pressure during isovolumic contraction, when LV pressure rises in preparation to eject its blood volume. Several factors affect these stages of diastole and are outline in table 1. Left ventricular chamber compliance is conspicuous by its influence on all three filling phases. Intrinsic factors that affect LV compliance include fibrotic or infiltrative diseases and myocardial hypertrophy. Extrinsic factors limiting LV compliance include pericardial disease and chronic lung disease. Heart rate influences diastolic function by limiting the time available for LV filling. The principal effect of tachycardia on diastole is the shortening and ultimate disappearance of diastasis. Other tachyarrhythmias such as atrial fibrillation produce inconsistent LV filling due to irregular intervals between LV contractions.

Since the four diastolic phases are generally described in terms of the relationship between LA and LV pressure, changes in diastolic function are reflected in typical Doppler transmitral flow patterns. However, as these patterns were recognized for different stages of diastolic dysfunction, limitations of transmitral flow to accurately represent LV filling became apparent. Notably dependence on preload was the principal disadvantage of transmitral flow in reflecting diastolic dysfunction. Pulmonary venous flow (PVF) also reflects changes in left atrial pressure and is a useful indicator of changes in left atrial pressure as a result of diastolic dysfunction. However, there are several limitations to the use of PVF as a sole indicator of diastolic dysfunction. These include the influence of preload, mitral regurgitation, dysrhythmias and difficulties in imaging pulmonary veins. Other indices less dependent on preload have been recognized and patterns defining diastolic dysfunction reported. These include Doppler-derived myocardial tissue velocity (tissue Doppler imaging, or TDI), and color M-mode propagation velocity (Vp) of early transmitral flow. Tissue velocity-based measurements represent a more preload insensitive index of diastolic function. Other aspects of diastolic function assessment include angle-independent

measures such as two-dimensional (2D) strain, strain rate, rotation and dyssynchrony. Acquisition techniques and patterns for some of these indices will be discussed.

Flow propagation velocity

The principal disadvantage of pulsed wave Doppler (PWD) is that it only detects flow at a single location determined by the sample volume. Color M-mode combines the superior temporal and spatial resolution of M-mode imaging with superimposed color coded velocity data. The result is a color coded display of velocities along a linear path extending from the LA to the LV apex across the MV (figure 2). The ME4CLAX view is the preferred view for this modality. A color window is selected with a narrow sector extending along the length of the image (from the tip of the LA to the tip of the LV apex) across the MV. The M-mode modality is then selected to obtain the color M-mode display. The color scale baseline is reduced to about 20 cm/s to enable visualization of the first aliasing velocity (bright yellow in figure 6). The slope of this first aliasing velocity column from the tips of the MV leaflets to the LV apex is the propagation velocity of early diastolic filling (Vp). Highest velocities are seen (as shades of darker yellow, figure 6) at or just below the level of the MV leaflets.

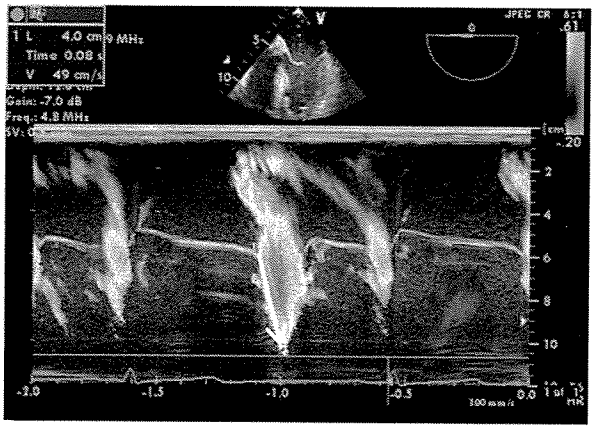


Figure 2: Color M-mode propagation velocity (Vp) of early diastolic flow. The slope of the first aliasing velocity

This parameter is also less affected by preload compared with transmitral flow and PFV.[3] In younger patients with normal LV function, the early diastolic suction effect may result in higher velocities near the LV apex rather than the MV. In patients with diastolic dysfunction and cardiomyopathies, the same column of blood moves at a slower pace towards the LV apex, resulting in a more obtuse slope and a Vp that is typically less than 45 cm/s. However, the eccentric direction of flow within the LV in advanced LV dysfunction may preclude accurate detection of a slope using this modality. Since determination of Vp depends on blood flow in line with the M-mode cursor, eccentrically directed flows result in loss of slope definition.

Tissue Doppler

Tissue Velocity Signals

While conventional Doppler techniques focus on high velocity, low amplitude signals of blood flow, TDI measures low velocity, high amplitude signals from myocardial tissue. It is therefore able to quantify myocardial velocity through time and provide information on systolic and diastolic tissue deformation. When measured at the lateral mitral annulus, these recordings are accurate, more reproducible and have higher temporal resolution than comparable modalities. There are three distinct signals from the lateral mitral annulus during the cardiac cycle – a systolic signal below the baseline directed away from the probe (s') and two diastolic signals, corresponding to early filling (e') and atrial contraction (a') (figure 3). Both diastolic signals are seen above the baseline, directed towards the probe, consistent with myocardial tissue motion in this phase.

The early diastolic myocardial velocity, or e' has several features that make it an attractive measurement for assessment of diastolic function. It is sensitive to myocardial ischemia, correlates with beta adrenergic receptor density, isovolumic relaxation constant (τ) and parameters of recoil. It is also less sensitive to preload conditions, which gives it an advantage over transmitral and pulmonary venous flow for diastolic function assessment. Atrial contraction produces the a' signal that indicates myocardial distension as a result of atrial filling.[4]

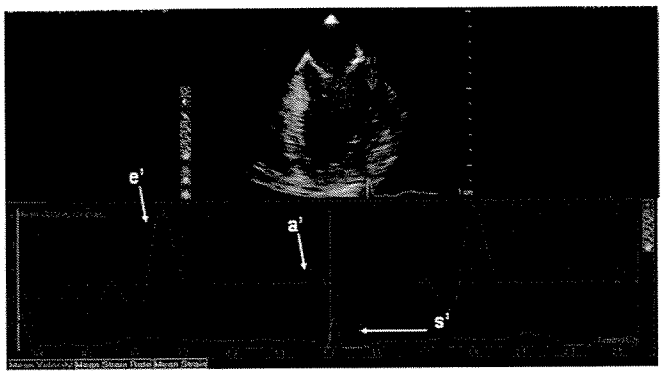
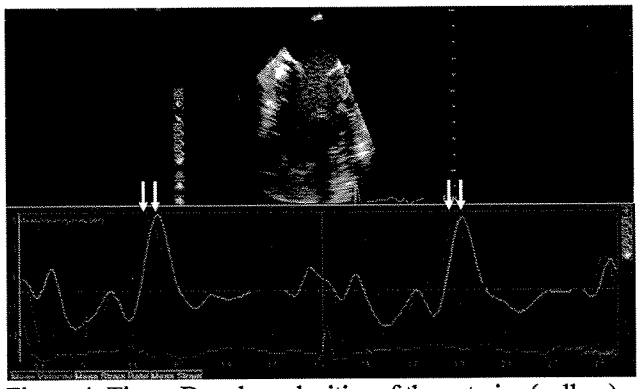


Figure 3: Typical diastolic tissue velocities obtained by tissue Doppler imaging of the lateral mitral annulus. See text for details)

In the early stages of diastolic dysfunction, the peak e' wave velocity decreases, while peak a' velocity shows a mild increase. With increase in left atrial pressures and progression of diastolic dysfunction to more restrictive physiology, the peak a' velocity decreases. However, the decrease in e' is more marked in these later stages of diastolic dysfunction. The decrease in peak e' velocity can be used as a reasonable marker of both, elevated left atrial pressure and left ventricular end diastolic pressure (LVEDP). Studies have shown that a peak e' of less than 8 cm/s is consistent with diastolic dysfunction. When considered along with transmitral early filling (or the E wave), the peak e' velocity can add diagnostic information, especially with normal ejection fraction. An E/e' ratio of >15 suggests elevated LVEDP, while a ratio of <8 is specific for normal diastolic function. Values between 15 and 8 indicate the need for other information. The European Society of Cardiology recommends assessment of left atrial and left ventricular volumes, transmitral or pulmonary venous flows, or evaluation of serum biomarkers to confirm the presence of HFNEF when the E/e' ratio is between 15 and 8.[5]

Diastolic Dyssynchrony

Advances in tissue Doppler and other echocardiographic techniques of tissue characterization, such as speckle tracking, have enabled a more detailed assessment of diastolic function. Tissue Doppler techniques have also evolved to allow simultaneous assessment of tissue velocities between different segments and within specific points in a segment. This feature also allows for the assessment of dyssynchronous diastolic relaxation patterns between segments. Subtle alterations in diastolic function may lead to regional relaxation abnormalities reflected only by assessment of segmental tissue velocities (figure 4).[6]


Figure 4: Tissue Doppler velocities of the anterior (yellow) and inferior (red) basal segments indicating dyssynchronous time to peak e' velocity between the two segments. See figure 3 for wave descriptions.

Diastolic Strain

An inherent limitation of tissue Doppler is its dependence on angle of interrogation similar to conventional Doppler. Thus, velocity and strain/strain rate can be assessed only in the longitudinal plane. The important contributions of circumferential and radial myocardial motion are therefore largely neglected. The advent of speckle tracking allows the myocardial tissue to be 'tracked' within a 2D image such that tissue properties can be assessed independent of angle of the ultrasound beam. This '2D functional imaging' allows angle-independent assessment of myocardial motion in multiple segments, including tissue velocity and strain/strain rate in longitudinal, circumferential and radial planes (figure 5).

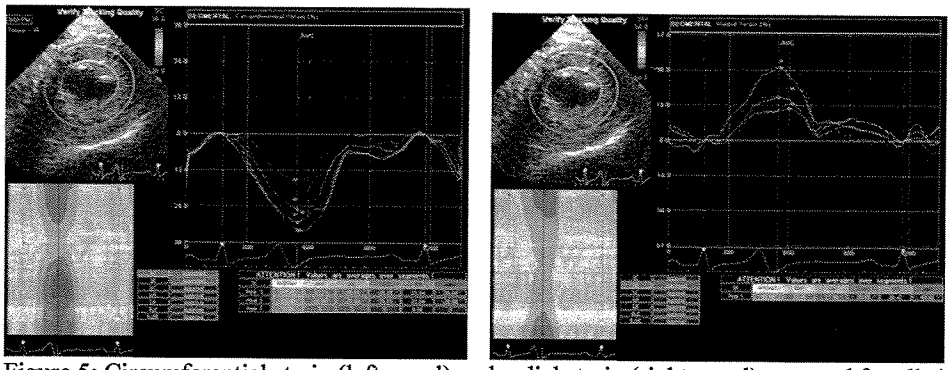


Figure 5: Circumferential strain (left panel) and radial strain (right panel) assessed for all six segments in the transgastric mid-short axis view. The software automatically calculates numerical values for global and segmental velocity, displacement, strain, and strain rate.

Diastolic 'Untwist'

The oblique spiral fibers of the heart impart a wringing motion at the base, also known as LV torsion. The systolic torsion is then followed by an 'untwisting' or recoil of torsion that occurs during the isovolumic relaxation time. During this early diastolic phase, the 'untwist' contributes to the diastolic suction effect that leads to rapid early LV filling. When seen from the apex, LV rotation is described as clockwise at the base and counterclockwise at the apex. Two values are considered important in the measurement of rotation – degree of rotation and rotational velocity. The reversal of both values in diastole (negative twist) represent the 'untwist' parameters that describe early LV diastolic function. Both, degree and velocity of negative twist are delayed in patients with LV hypertrophy and may be early indicators of diastolic relaxation abnormality.[7] This modality may also help provide further insight into subtle changes in diastolic function that are seen in the immediate postoperative period.

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Tissue Doppler and Strain

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Learning objectives:

1. Understand the techniques for acquiring Doppler tissue imaging, strain, and strain rate
2. Be able to describe applications for Dopper tissue imaging, strain, and strain rate in the evaluation of myocardial performance

The traditional echocardiographic assessment of global and regional left ventricular (LV) function is based on 2D and Doppler methods examining cardiac motion and blood flow velocities. However, there are many limitations: fractional area change and ejection fraction (EF) are influenced by load and do not necessarily express myocardial contractility, Doppler calculation of stroke volume (SV) is prone to measurement errors, rate of of LV pressure rise (LV dP/dt) requires the presence of mitral regurgitation, while visualization of the endocardial excursion may be suboptimal, and can not differentiate an actively contracting from a passively drawn segment if the epicardium is not seen. Technological advances in signal processing make it possible to directly measure myocardial velocity (tissue echocardiography) and deformation (strain) in real time, and describe myocardial performance more accurately.

Anatomic and physiologic background – The LV longitudinal motion

During LV systole, the myocardium shortens in the long axis (the mitral annulus [MA] descends towards the relatively stationary apex), thickens in the short axis, while the base and the apex rotate in opposite directions. Since muscle is incompressible, the longitudinal deformation of a tissue segment will correspond inversely to the changes in thickness. The anatomic background of this motion lies in the myocardial architecture. The epicardium and subendocardium have longitudinal fibers (twisting from the base to the apex in a clockwise and counter-clockwise orientation respectively), and the mid-myocardium contains circumferential fibers. During systole, tension develops first in the longitudinal fibers, whereas thickening of the circumferential fibers predominate during ejection. In diastole, the circumferential fibers predominate in the early LV wall motion, while the longitudinal fibers predominate at atrial contraction. Therefore, monitoring the heart motion along the longitudinal axis has the potential to offer valuable information about heart function.[Fig 1]

Myocardial velocity: Principles of tissue Doppler imaging

Conventional Doppler records blood flow velocity. Tissue Doppler imaging (TDI) maps tissue velocity. Tissue motion creates Doppler shifts that are stronger (approximately 40 dB higher in amplitude) and significantly lower in velocity (< 25 cm/s) than those from blood flow. Reducing gain amplification, and bypassing the high-pass wall filters enables

tissue imaging. TDI requires a high frame (>100 FPS), by using a narrow image sector and selecting the appropriate velocity scale.

Tissue Doppler imaging modalities and measurements

Spectral TDI provides on-line measurements of peak myocardial velocities and time intervals with excellent temporal resolution. The myocardial area of interest is placed in the center of the ultrasound beam for parallel alignment of motion plane with the cursor. TDI velocities are recorded from a sample volume (2-5 mm), and are positive if moving toward, and negative if moving away from the transducer. The Nyquist limit should be < 20-30 cm/s, with minimum gain and low wall filter settings, and the sweep speed should be set at 50-100 mm/s. Limitations of TDI are: (i) inability to differentiate between actively contracting and passively drawn tissue (tethering), (ii) inability to analyze separately the different myocardial layers (i.e., sub- from epicardium), and (iii) Doppler velocities are angle-dependent.

- The spectral TDI velocity signal has 3 main components: systolic (Sm), early (Em) and late diastolic (Am).[Fig 2] Sm may show 2 components: S'_1, during isovolumic contraction (IC), and S'_2, during systolic ejection. The diastolic velocities are similar in appearance and timing to the trans-mitral flow velocities. The late (Am) diastolic velocity may not be evident in atrial fibrillation or tachycardia. An isovolumic relaxation (IR) velocity can be seen before the Em. Measurements include peak velocities (cm/s) and the ratio Em/Am (%). When recorded from the MA, the velocities receive the subscript "a", i.e., Sa, Ea, Aa. (Figs 1 and 2)
- TDI velocities can be recorded with transesophageal echocardiography (TEE), from the following walls: (i) infero-septal, antero-lateral, inferior, and infero-lateral in the ME LV views and antero-septal in the deep TG LV LAX view, representing LV longitudinal contraction and relaxation, (ii) inferior and anterior in the TG mid SAX view, (iii) right ventricular (RV) free wall lateral to tricuspid annulus in the ME 4C or the modified deep TG RV LAX view at ~130° (inflow part of the RV), and (iv) RV outflow tract at the level of the pulmonic valve, in the modified deep TG RV LAX view at ~70°.
- Spectral TDI velocities are influenced by cardiac translation and pericardiotomy. When recorded from the LV base, lateral to the MA, these influences are minimized. TDI velocities are heterogeneous: Sm is higher in the LV basal than in the mid or apical segments, and in lateral and inferior than anterior or septal segments. Em is greater at the base than in the mid LV, while Am has a more uniform distribution. TDE velocities decrease with aging. Normal values for spectral TDI velocities (cm/s ± 1 SD) for the basal segments of the LV in *awake* patients are shown in the table below.

	Sm	Em	Am	Em/Am
Lateral	10.6 ± 2.3	13.3 ± 3.3	11.3 ± 2.9	1.5 ± 0.6
Septal	9.9 ± 1.7	11.5 ± 2.6	9.5 ± 2.4	1.0 ± 0.7
Anterior	9.2 ± 1.8	11.7 ± 3.4	10.3 ± 2.9	1.2 ± 0.7
Inferior	10.4 ± 2.5	14.3 ± 3.6	11.6 ± 2.6	1.3 ± 0.7

In **color TDI,** the velocities are displayed as a parametric color image (a color Doppler B-mode), in which each pixel represents the velocity relative to the transducer, and are encoded with red if moving toward, and blue if moving away from, the transducer. The images are digitally acquired and stored, and mean myocardial velocities are extracted off-line. Different myocardial layers can be separately analyzed, since the

sample volume is positioned post-processing. Conventional or anatomically curved M-mode color TDI provides adequate temporal resolution (10-100 ms) to analyze cardiac events, and display segmental asynchrony between different myocardial segments (i.e., basal vs mid vs apical). In a TG view, the velocity gradient between endocardium (moves faster) and epicardium reflects the rate of change in wall thickness and is equivalent to strain rate (see below).

Clinical applications of TDI

- EF estimation:

TDI does not depend on endocardial border definition. Tissue velocities may estimate LV function better than the other methods. In a population of healthy and diseased subjects, Sm (a 6- or 2-segment average from the LV base in apical 4C view) correlated closely with LV EF. An Sm >5.4 cm/s had a favorable sensitivity and specificity for rapidly identifying EF within the normal range (%EF = 8.2 × Sm + 3%, Sm: 6-segment average). This relationship between Sm and LVEF was not significantly affected by the presence of regional wall motion abnormalities. If Sm at the infarct site is > 7.6 cm/s and the time interval from Q-ECG to peak of Sm is < 200 ms, then %EF is > 50%.

- Ischemia detection:

The echocardiographic evaluation of coronary disease is assessed by the visual analysis of regional wall motion abnormalities (RWMA), based on the detection of alterations in radial motion and systolic thickening. The qualitative evaluation of RWMA is unable to resolve short delays in regional contraction and may fail to identify ischemia-induced myocardial dysfunction. Furthermore, changes occur earlier in the long than the short axis. In an animal model, a significant reduction of Sm and an early decrease of Em/Am were detected within 5 s of acute occlusion of the left anterior descending artery. Patients with myocardial infarct tend to have significantly reduced Sm in the non-infarcted walls as well. Em is reduced in the infarction sites, and is affected by ischemia more than Am, even in the presence of intact systolic function. Non-ischemic segments have an Em/Am >1. An Em/Am <1 is sensitive for ischemia, and is found in akinetic and hypokinetic segments. The TDI Em/Am ratio is dimensionless and is not Doppler-angle dependent.

- Diastolic function and estimation of filling pressures:

Em is related to LV relaxation and elastic recoil and correlates inversely with peak negative dP/dt and LV end-systolic volumes. Em is higher in lateral, inferior and posterior segments than in the anterior and septal segments, and a value >12.5 cm/s is associated with normal diastolic function, particularly when measured during a Valsalva maneuver. Compared to the load-dependent trans-mitral (TMF) E-wave, Em at the lateral LV base is relatively preload independent. Therefore, the effects of preload on the LV filling can be corrected by a ratio of TMF-E/Em, and reveal underlying relaxation abnormalities. The diastolic function is abnormal if E/Em >10, and can be utilized to further characterize the patient with "pseudonormal" filling pattern. E/Em > 10 is associated with wedge pressure > 15 mmHg (mean capillary wedge pressure = 1.9 + (1.24 × E/Em). E/Em >15 was highly specific for elevated, and E/Em <8 was highly sensitive for normal LA pressures.

- Heart disease:

Chronic volume overload influences Em. Patients with mitral regurgitation (MR) may have higher Em velocities than patients with aortic regurgitation. TDI can detect

subclinical dysfunction in minimally symptomatic MR patients with normal EF. If the contractile reserve is intact (%EF increase >5% during exercise), the resting Sm was higher (10 ± 2 cm/s) than if impaired (8 ± 2 cm/s). TDI can detect subclinical dysfunction in patients with cardiomyopathy, even when conventional echocardiography is normal. A mean Sm <9 cm/s differentiates pathologic from physiologic hypertrophy. In heart failure patients, TDI can offer important prognostic information: in a 2-year follow-up of 518 patients (165 normal), Sm ≤ 3 cm/s, Em ≤3 cm/s, Am ≤4 cm/s, and E/Em > 20 independently identified those at risk of cardiac death.
- Right ventricular function:
TDI imaging of the RV inflow provides quickly, on-line evaluation of the RV function. An Sm <11.5 cm/s predicts RV dysfunction (%EF <45%).

Myocardial deformation: Strain and Strain Rate
Strain (ε, %) is defined as the deformation of an object after application of force (stress), normalized to its original shape (or length, L_o): $\varepsilon = (L - L_o) / L_o$. In a one-dimensional object, the only possible deformation is shortening (negative ε) and lengthening (positive ε). (Fig 3, 4) An example of one-dimensional ε is wall thickening (WT), measured in the anterior or inferior wall segment in the TG SAX view: $WT = (L_{ED} - L_{ES}) / L_{ED}$.
Lagrangian strain is the instantaneous ε, in relation to time (t): $\varepsilon(t) = [L(t) - L(t_o)] / L(t_o)$, where $L(t_o) = L_o$.
Natural strain is the deformation when the reference value (L_o) is continuously changing. For a 2D object, deformation occurs along both the x axis and y axis (*normal* strain: the motion is normal to the borders of the object), and parallel to the borders of the object (*shear* strain). For a 3D object, such as a myocardial segment, there are three *normal* strains (along the x, y, z axes), and six *shear* strains (along the axes combinations). For the evaluating of the myocardium, the natural strain should be measured, since the deformations are independent of the initial length L_o. During ventricular contraction, negative ε will be recorded from the muscle shortening in the longitudinal and circumferential dimensions (with the cursor lined up with the heart long axis), and positive ε from the muscle thickening in the radial direction (with the cursor lined with the short axis). Normal values (in the same segment): longitudinal ε: -25 ± 7% (range: 15%-25%), radial ε: -57 ± 11% (range: 50%-70%).
Strain Rate (SR, $\hat{S}$, s^{-1}) is the speed of deformation. If an object had a 30% total deformation over 2 sec, then SR is $0.30/2 = 0.15\ s^{-1}$ (the object lengthens by 15% every sec). In most biological tissues the relationship between stress and strain is not linear. Normal values: longitudinal SR: $-1.9 \pm 0.7\ s^{-1}$, radial SR: $-3.7 \pm 0.9\ s^{-1}$. (Fig 5)
Strain represents *local* myocardial shortening and lengthening, while TDI measure motion velocity relative to the transducer. Therefore, SR and ε are less sensitive to segment tethering and cardiac translation. The echocardiographic strain has been validated with mechanical strain, measured with sonomicrometry and magnetic resonance imaging. Myocardial deformation is the result of the complex interaction of intrinsic contractile force and extrinsic loading conditions applied to a tissue with viable elastic properties. Changes in preload, afterload and myocardial stiffness are important determinants of the pattern and magnitude of myocardial deformation. Thus, contractility can not be measured directly with SR and ε. If the contractile function is preserved, systolic ε will increase with increasing preload and stroke volume, and will decrease with

increasing ventricular size and afterload. Peak SR correlates well with LV dP/dt. Myocardial deformation can be measured in radial (transmural, basal and mid inferior and anterior segments in the TG SAX view, and basal and mid inferolateral in the TG 2C view), longitudinal (all six walls using the ME views), and circumferential (septal and lateral segments in the TG SAX view) direction. SR is age-dependent. Longitudinal SRs are relatively heterogeneously distributed from base to apex, and are approximately half of the radial SRs. A transmural gradient exists in normal myocardium, with higher SRs in the subendocardial layer. The RV free wall SR is higher than the LV SR.

Echocardiographic techniques for measurement of SR and ε

- **Cross-Correlation method:** it measures the similarity of two signals as a function of their relative phase. The spatial gradient is used to estimate ε, and from that, its temporal derivative (SR), is calculated.
- **Velocity-Gradient method:** if L in the strain equation is divided by (t), then SR can be calculated from velocities (v): $SR = [v - v_o] / v_o$. The velocity of myocardial motion is recorded by color TDI techniques, and the following *regional* parameters are derived from a sample volume: TDI velocities (Fig 2) and tissue displacement (time integration of TDI velocities), SR (the spatial gradient of velocities) and ε (time integration of SR). An anatomically curved M-mode line allows investigation of several segments simultaneously.
- **2D method (2DSE):** this technique is not based on Doppler. The myocardial deformation is determined from continuous frame-by-frame tracking of a small block of speckles ("acoustic markers"). The appearance of these markers is considered to be relatively stable between subsequent image frames, and a change in their position is assumed to follow tissue motion. 2D ε and SR are calculated from the displacement and rate of displacement of each marker. 2DSE can measure global deformation, has better spatial resolution and requires a low frame rate (~60 per sec). 2DSE longitudinal ε has a better correlation with sonomicrometry than circumferential ε. Radial ε can not be measured with 2DSE.

Limitations of SR echocardiography

- Measurement of SR is prone to noise. A 5- to 10-mm sample volume is a reasonable trade-off between high spatial resolution and low noise. Analysis of ε and 2DSE are less prone to noise.
- All strain methods (but not 2DSE) are limited by the Doppler angle. In clinical practice, this angle dependency does not probably have an effect on the timing of specific events or on the profile of the curves.
- Recordings should be done with second harmonic imaging to optimize tissue visualization.
- Longitudinal and circumferential deformations have opposite signs than radial deformation, and deviation from one direction to another will consequently affect the SR values when measured with the velocity-gradient method. Furthermore, measurements are done along a single ultrasound scan axis/dimension, and provide only one-dimensional estimate of myocardial deformation, even when the sample volume is "anchored" within a segment. Not all strain components (radial, longitudinal and

circumferential) can be measured in all LV segments. To improve accuracy a frame rate of >100 frames/s is required.

Clinical applications of strain echocardiography

- Ischemia evaluation

Ischemic myocardial changes can be recorded from all segments, much earlier than with visual estimation or TDI velocities. The regional SRs have been validated to correlate with those obtained from sonomicrometry in acute coronary ischemia. SR and ε have the potential to discriminate acute from chronic ischemia and myocardial infarction: (i) the homogeneous distribution of systolic SR is lost in ischemia and infarction, (ii) peak SR is reduced or reversed within seconds after coronary artery occlusion, (iii) early systolic *thinning* and a delay in the onset and degree of systolic *thickening* appear in a flow-related and progressive degree of acute ischemia, (iv) an abnormal, ischemia-induced delay in the myocardial deformation occurring during IR, and extending well into the early filling period, is called post-systolic thickening (PST) if recorded in the radial direction, or post-systolic shortening (PSS) if recorded in the longitudinal direction. Small amplitudes of PST are sometimes present in normal myocardium but are lower than systolic ε. With increasing ischemia, there is a progressive reduction in maximal SR and ε with a concomitant increase in PST (> 25%-35% of systolic ε). The extent of myocardium exhibiting PST approximates the extent of myocardium at ischemic risk, (vi) an increase of the time to onset of relaxation (T_R) >20% identifies new RWMA with high sensitivity and specificity, in a more reproducible way, and with less noise.
Significant changes in regional deformation during acute ischemia occur even in segments that do not appear to deteriorate by visual evaluation. Using 2DSE, a global 2D-strain cutoff value of -21% and a global 2D-SR value of -0.9 s^{-1} have good sensitivity and specificity for detection of myocardial infarction.

- Strain echocardiography during low-dose dobutamine challenge allows the differentiation of normal from ischemic (with or without flow reserve) and infracted tissue. At rest, stunned (with present flow reserve), acutely ischemic tissue and non-transmural infarction are associated with reduced ε and SR, and presence of PST. Transmural infarction is associated with lower ε and SR and less PST. Low-dose dobutamine (10-15 mcg/kg/min) increases ε and SR and reduces PST in stunned myocardium, but non-transmural infarcts show only a transient increase of SR (an optimal cutoff of SR increase of >0.23 s^{-1} predicts viability), no change of ε, and increased PST. Acutely ischemic tissue deteriorates and transmural infarction remains unchanged. The lack or response of PST during dobutamine challenge, shows that PST is a passive phenomenon in transmural infarctions, and not a direct marker of segmental viability.

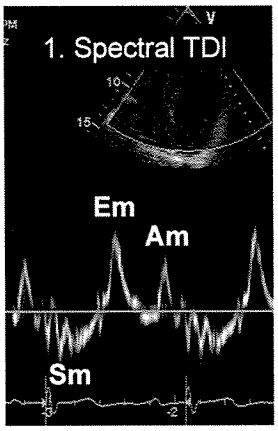
A step-by-step evaluation of ischemic segments has been suggested, based on the above: (i) visual inspection for synchronicity, (ii) TDI if there is a regional wall motion abnormality, and challenge with low-dose dobutamine to investigate the systolic velocity response, (iii) SR and ε to further define the ischemic substrate. In hearts with conduction or rhythm abnormalities, use SR echocardiography first. In the setting of global disease, in which the site specificity of SR imaging is not required, strain echocardiography may not offer a clear advantage over TDI.

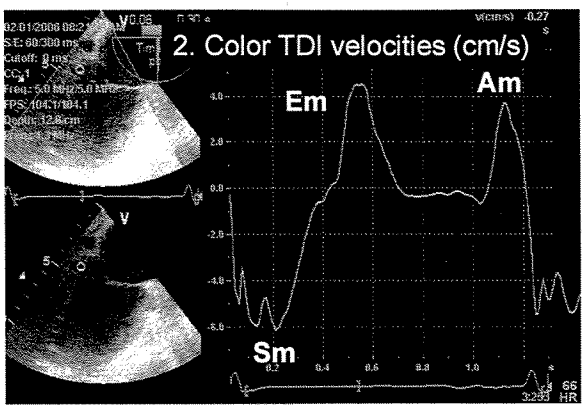
- Valvular heart disease
Regional SR and ε can differentiate between aortic stenosis patients with and without coronary artery disease. In regurgitant lesions with intact regional contractility, regional systolic and diastolic strain indices increase with increased preload.
- Cardiomyopathies
Systolic SR was lower in restrictive cardiomyopathy than in constrictive cardiomyopathy, and in hypertrophic cardiomyopathy than in athletes, hypertensive patients or healthy subjects.
- Diastolic function
Differences in SR during the initial phases of diastole appear to identify early changes in regional myocardial function (in the absence of systolic and gross morphological changes). Regional abnormalities in diastolic function (which may indicate the presence of regional disease or may be caused by aging) are shown by changes in TDI-Em (impaired relaxation) and regional ε curves (changes in compliance).
- Cardiac synchronization therapy (CRT)
Appropriate CRT can acutely improve regional SR and reduce the regional delay between QRS onset and peak ε, even without changes in systolic TDI velocities.
- Right ventricular function
The RV SR and ε profiles are difficult to measure because the wall is thin. There is heterogeneity between the inflow part (lowest values) and outflow tract (highest values). Peak SR correlates with systolic pulmonary artery pressure. In pulmonic stenosis, ε is homogeneously reduced in relation to the RV systolic pressure increase, while it is increased in pulmonary hypertension (increased preload and afterload) and atria septal defect (preload increase).

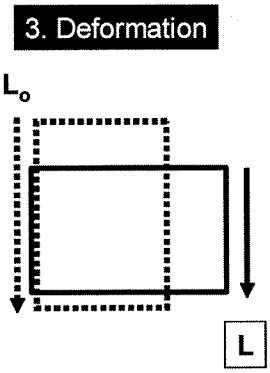
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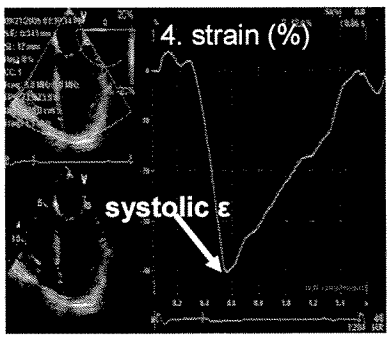
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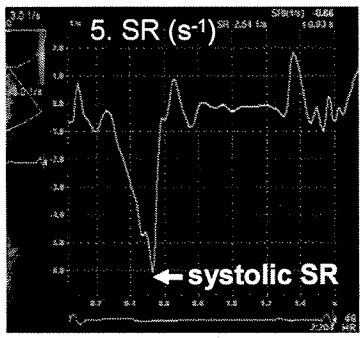
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Three-Dimensional Echocardiography

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Introduction

Three-dimensional echocardiography (3-DE) refers to the technique of visualizing the heart and its structures and flows in the three-dimensional format either in real-time or after reconstruction from multiple two dimensional images. The concept of 3-DE was first introduced in the early 1970s.[1,2] Only recently, has it gained attention for its utility in the perioperative environment. This is primarily due to faster acquisition times and better methods to analyze the data obtained for patient management and surgical decision-making.

Today, perioperative 3-DE can be performed by a transthoracic (TTE), transesophageal, or epicardial approach.[1] Two of the most common intraoperative methods of 3-DE include transesophageal rotational-gated acquisition and epicardial real-time 3-D echocardiography (RT3DE). Briefly, transesophageal rotational-gated acquisition involves acquiring rotational 2-D slices from 0 to 180 degrees with electrocardiographic and respiratory gating. Gating reduces motion artifact in the reconstructed 3-D image. The 60 to 90 two-D echocardiographic slices are then reconstructed with interpolation of the 2-D data sets to fill in the gaps between imaging planes to form a final 3-D image. The 3-D data set can then be displayed in a variety of formats.[3,4] An infinite number of cutting planes can be displayed as well as quantitative data analysis and color flow Doppler to assist with surgical decision-making. RT3DE compared with conventional 3-D reconstruction, permits faster acquisition without a dependency on the electrocardiogram or respiratory gating, and allows simultaneous visualization of orthogonal planes with 2-D resolution.[5]

Three-DE has not been embraced by perioperative echocardiographers because of the long acquisition times needed for 3-D TEE reconstruction and off-line analysis of data sets. In the past year, a RT3-D TEE transducer has been developed (Philips Medical Systems Inc., Andover, MA) to allow real-time acquisition and on-line display of 3-D images, in addition to performing complete 2-D and Doppler exams. A recent abstract looking at the feasibility of intraoperative RT3-D TEE showed that the new technology provides practical and unique echocardiographic imaging planes for identifying intra-cardiac pathology. Compared to conventional 2-D imaging and gated 3-D reconstruction echocardiographic techniques, RT3-D TEE may permit a more practical and comprehensive echocardiographic evaluation to facilitate perioperative surgical planning.[6]

We are only beginning to realize how 3-DE can improve our understanding of ventricular and valve function. For example, 3-DE is providing valuable insight in the complex anatomic relationships that 2-DE fails to adequately image, such as the mitral annulus and its saddle shape.[7,8,9] In addition, 3-DE provides accurate quantification of left and right ventricular volumes, mass, and ejection fractions without making geometric assumptions as in 2-DE.[10] Furthermore, there is improved visualization of spatial relations and 3-DE provides unique "en face" views and surgical views not previously obtainable by 2-DE[10]. In a recent study of 106 patients, RT3DE provided new finding or a useful anatomic perspective in 25% of the studies when compared to conventional 2-DE.[11] This was especially true in patients with mitral valve disease and congenital heart disease. To date, much of the published literature on 3-DE focuses on ventricular function and volume analysis, mitral valve structure and function, congenital abnormalities such as atrial septal defects and ventricular septal defects, and intracardiac masses.

Ventricular Analysis

The utility of 3-DE in the evaluation of ventricular function has been looked at in several recent studies. Qin et al used RT3DE to quantitatively assess changes in LV volume and function after LV reconstruction surgery in patients with ischemic cardiomyopathy.[12] Shiota and McCarthy determined that 3-DE is valuable in facilitating the communication between echocardiographers and cardiac surgeons in volume reduction surgery for end-stage ischemic heart disease.[13] Arai et al determined that RT3DE allows convenient and accurate estimation of LV volume and ejection fraction in patients with wall motion abnormalities.[14] Three-DE is advantageous in the patients who have ventricles with complex geometry since it does not rely on geometric assumptions to make these calculations.[10] The usefulness of RT3DE is also being evaluated in patients with obstructive hypertrophic cardiomyopathy.[15,16,17] RT3DE is being used to determine the exact location of the systolic anterior motion of the mitral leaflet, the extent of mitral leaflet to septal contact, and the precise area of the left ventricular outflow tract obstruction.

Mitral Valve

Intraoperative surgical planning depends on proper evaluation of the mitral valve apparatus. The feasibility, accuracy and value of intraoperative 3-DE in valve surgery was studied by Abraham et al.[18] Intraoperative 2-D and 3-D reconstruction TEE examinations were performed on 60 patients undergoing valve surgery. Three-D acquisitions were completed in 87% of the patients within a mean acquisition time of 2.8 ± 0.2 minutes and reconstruction time within 8.6 ± 0.7 minutes. Three-D echocardiography detected all salient valve morphological pathology (leaflet perforations, fenestrations and masses), which was subsequently confirmed on pathological examination in 84% of the patients. In addition, intraoperative 3-D TEE provided new additional information not obtained by 2-D TEE in 15 patients (25%), and in 1 case influenced the surgeon's decision to perform a valve repair rather than a replacement. Furthermore, intraoperative 3-DE provided worthwhile and complimentary anatomic information that explained the mechanism of valve dysfunction demonstrated by 2-D imaging and color flow Doppler.

The ability of 3-DE to render new views not previously obtainable by 2-DE, such as the "en face" and surgical views of the mitral valve, provide new insight and possible better communication to the surgeon. Ahmed et al evaluated the potential utility of 3-D TEE in identifying individual MV scallop prolapse in 36 adult patients undergoing surgical correction.[19] Perfect correlation between 3-D TEE and surgical findings was noted in 78% of the patients. Similarly, De Castro et al demonstrated superior concordance between intraoperative 3-D TEE surgical identification of prolapsing anterior and posterior MV scallops compared to 2-D TEE.[20]

Being able to "cut" the mitral valve in any plane from both atrial and ventricular views adds a new dimension to its evaluation. In studies comparing 2-D vs 3-D TEE for measuring MV area and volume in patients with stenosis undergoing percutaneous MV balloon valvuloplasty (PMTV), 3-D TEE enabled a better description of valve anatomy including the identification of commissural splitting and leaflet tears post PTMV.[21,22,23] Two studies have shown that 3-DE of the mitral valve allows direct visualization and accurate planimetry of the regurgitant orifice area in patients with mitral regurgitation when compared to the proximal flow convergence method.[24,25]

Three-DE with color flow Doppler was evaluated by Sugeng et al.[26] The study looked at 46 patients with mitral regurgitation. 3-D color flow Doppler was particularly helpful in patients with paravalvular leaks and eccentric jets. Mukhtari et al reported on two cases where 3-DE color flow Doppler was more accurate in defining the exact site of prosthetic aortic valve dehiscence and communication with an abscess cavity.[27] De Simone et al found that calculated jet volumes from 3-D Doppler reconstruction in patients with eccentric mitral regurgitation significantly correlated with angiography, regurgitant fraction, and regurgitant volume.[28]

Aortic, Pulmonary, and Tricuspid Valves

Pulmonary and tricuspid valves in the past have been difficult to image, but 3-DE may provide better evaluation of these valves in the future. Handke et al reports on a new 3-DE system that utilizes radiofrequency data that enables high-resolution imaging of cardiac movements.[29] This enables better qualitative and quantitative analysis of valve structure, such as aortic valves, which in the past have been more difficult to see with 3-DE because of its thinner leaflets.

Congenital Pathology

Three-DE is being used to evaluate congenital heart pathology as well, including atrial septal defects and ventricular septal defects. The ability to look at the defect "en face" from both the left and right atrial views and the left and right ventricular views helps to delineate the borders and size of the defect. Lange et al compared preoperative 2-D and 3-D TTE evaluations with intraoperative findings in 15 patients with atrioventricular septal defect morphology.[30] Three-DE reconstruction provided superior imaging of the MV and tricuspid valve function, a more precise description of primum atrial septal defect (ASD) size, secundum ASD fenestrations and ventricular septal defect (VSD) size. Three-DE measurement of ASD size has correlated well with findings obtained intraoperatively or during transcatheter closure.[31] A similar degree of accuracy for 3-DE evaluation of VSD size prior to closure has also been demonstrated.[32] Two case reports have demonstrated the accuracy of 3-DE in imaging post-infarct VSD's and pseudoaneuryms.[33]

Intracardiac Masses

The evaluation of intracardiac masses appears to be improved by 3-DE. Borges et al states that 3-DE yields important additional clinical information and improves the operative planning.[34] The complimentary information provided by 2-DE and 3-DE was able to demonstrate the shape, dimensions, location, origin, three-dimensional movement, and valve involvement of cardiac tumors. There are multiple reports of 3-DE further defining ventricular thrombi and assisting in surgical planning.[35,36,37]

Limitations

Although 3-DE has shown promise it still has drawbacks that need to be considered. Three-D gated reconstruction requires additional time to acquire and reconstruct the images. Any type of motion artifact in the 2-D images prior to reconstruction will make the 3-D data set suboptimal.[38] Patients with atrial fibrillation or an irregularity in their RR interval will prolong data acquisition and make the 3-D reconstruction of poorer quality. Epicardial RT3DE has its own limitations. Epicardial scanning takes practice to be able to visualize the structures you want to see in RT3D. A large water or gel step-off is needed to visualize structures close to the surface of the heart. If the surgeon is performing the scanning, communication is very important to acquire the images. RT3D TEE is a new technology. It ability to visualize near-field structures such as the left atrium, mitral valve and left ventricle appears superior to far-field structures such as the tricuspid and pulmonic valves.[6] Real-time 3-D images have slower frame-rates compared to traditional 2-D imaging.

There is a learning curve to data acquisition and reconstruction so that the structure needed for analysis is optimally displayed. Manipulation of gain and threshold on pre and post image processing is very important to accurately image various structures and avoid 3-D dropout, which can be misleading when evaluating cardiac structures.

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NOTES

Echocardiographic Evaluation of Cardiomyopathies

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Introduction

Cardiomyopathy is generally defined as a "disease of the myocardium associated with cardiac dysfunction".[1] *Primary cardiomyopathies* are divided into three major classifications: a) dilated cardiomyopathy (DCM), b) hypertrophic cardiomyopathy (HCM), and c) restrictive (or infiltrative) cardiomyopathy (RICM). Although there are functional and clinical overlaps, each of the primary cardiomyopathies has distinctive morphological and functional characteristics.

The incidence of cardiomyopathy is less than 1% in the general population, with DCM representing the vast majority of cases.[2] Hypertrophic cardiomyopathy is less prevalent while RICM is the least common. Accurate diagnostic assessment of patients suspected of having a cardiomyopathy is important to establish prognosis and to institute appropriate treatment. This talk will focus on salient echocardiographic features of primary cardiomyopathies, but may include discussion and description of none-primary cardiomyopathies for completeness.

Dilated Cardiomyopathy

Etiology and Clinical Presentation

Dilated cardiomyopathy (DCM) accounts for 60% of all cardiomyopathies and is characterized by progressive myocyte hypertrophy, dilation and contractile dysfunction of one or both ventricles.[3] Although ventricular wall thickness can be increased, the degree of hypertrophy is proportionally less compared to the amount of dilatation.[4] The development of left ventricular (LV) hypertrophy is initially beneficial in reducing systolic wall stress, a major determinant of myocardial oxygen consumption. However, wall stress is never fully normalized and eventually stimulates LV remodeling, resulting in a reduced ejection fraction (EF) as the ventricle continues to dilate and assume a spherical shape.[5] DCM is defined by increased end-systolic and end-diastolic volumes and a reduced LV EF (< 45%). Furthermore, mitral (MV) and tricuspid (TV) regurgitation may be present in association with increased ventricular volume and/or pressure load. Cardiac thrombi may also develop in associated with reduced cardiac function, and are most commonly found in the LV apex or left atrial appendage (LAA).

Many patients with DCM may be asymptomatic, however, most patients range in age between 18 and 50 years, with progressive deteriorating DCM include clinical signs and symptoms all of which are consistent with LV heart failure.[6] The right ventricle (RV) may be independently involved in rare cases of a familial form of DCM, however RV failure is usually a later and more ominous consequence of primary LV failure, and is usually associated with a particularly poor prognosis.[7]

As many as fifty percent of patients die within 2 years. Five-year survival following the initial diagnosis has been reported in the 50-75% range depending upon the initiation of therapy,[8] extent of cardiac remodeling and dysfunction, and advanced age.[9,10] About 25% of patients with recent onset DCM improve spontaneously.[11] Patients with

DCM and advanced CHF with LV end-diastolic diameters > 4 cm/m^2 body surface area have twice the 1-year mortality rate compared to those patients with less significant ventricular dilatation.[10]

Echocardiographic Evaluation

Classic 2-D echocardiographic features of DCM include the presence of increased systolic and diastolic (>7-8 cm) LV dimensions, reduction of LVEF (<45%), and development of a spherical configuration.[12] Although DCM is defined by the presence of significant systolic dysfunction, concurrent diastolic dysfunction is common and may manifest anywhere within the full spectrum of severity from impaired relaxation to restriction. Symptoms of CHF in patients with DCM appear to be related to the severity of diastolic dysfunction.[13,14]

The LV wall can vary from normal to increased thickness; however, relative wall thickness (i.e. the ratio of end-diastolic wall thickness to end-diastolic cavity radius) is severely diminished. Although not pathognomonic, LV and/or RV wall motion tend to be *symmetrically and globally* reduced in patients with DCM compared to the typical *segmental and focal* wall motion abnormalities more commonly associated with ischemic heart disease and coronary artery narrowing. Dobutamine stress echocardiography may RWMA associated with CAD, differentiating it from patients with idiopathic DCM.[7,15] It is important to make this distinction since patients with ischemic cardiomyopathy may experience significant improvement in functional capacity with coronary revascularization.

Ventricular dilatation associated with DCM may produce functional atrial-ventricular valve incompetence. Incomplete closure of the MV and TV may develop due to annular dilatation, abnormal alignment of the papillary muscles related to the development of ventricular sphericity, and apical displacement of the coaptation point which increases tension on the leaflets (i.e. "apical tenting").[16,17]

The risk of intracavitary thrombi, due to flow stasis, are higher for patients with DCM. Thrombi develop more commonly in the LV compared to the LA.[18]

Hypertrophic Cardiomyopathy

Etiology and Pathophysiology

The classification of hypertrophic cardiomyopathy (HCM) has been simplified to reflect the pathophysiology and to accommodate the varied patterns of hypertrophy. While diastolic dysfunction occurs in almost all individuals with HCM, only 25% experience a dynamic, intermittent or episodic obstruction to ventricular systolic outflow.[19,20] Patients are therefore subdivided into two related but distinct groups: 1) hypertrophic cardiomyopathy (HCM); and 2) hypertrophic obstructive cardiomyopathy (HOCM), which includes those with obstruction to ventricular systolic outflow.

Hypertrophic cardiomyopathy (HCM/HOCM) is defined as an abnormal thickening of the myocardium without chamber dilation, and in the absence of a demonstrable cause (e.g. aortic stenosis [AS], systemic hypertension). The incidence is approximately 0.2% in the general population, but reporting may vary depending on the referral patterns of the institution, and the diagnostic criteria.[19,20]

The age of onset, morphology, and pathophysiology of HCM/HOCM varies greatly. More commonly, HCM/HOCM presents in young adulthood from the second to fifth decades, and is characterized by a diffuse or asymmetric ventricular hypertrophy.

Presentation of HCM/HOCM in older patients (≥ 65 years old) is becoming increasingly more recognized.[21,22] Although commonly ascribed to HCM/HOCM, asymmetric hypertrophy has also been reported as an adaptive response to AS, systemic hypertension, and in certain congenital cardiac anomalies.

Hypertrophic cardiomyopathy is defined by the presence of LV hypertrophy (> 11 mm thickness), which occurs disproportionately in the ventricular septum by a ratio of > 1.3:1.0 relative to the measured free wall thickness.[20] However, different patterns of hypertrophy have been reported including isolated proximal basal septal hypertrophy ("septal bulge"),[21,22] posterior wall hypertrophy,[23] concentric or diffuse hypertrophy,[24] and RV hypertrophy in a small number of cases. Four types of hypertrophic cardiomyopathy have been described: Type I – hypertrophy limited to the anterior septum; Type II – hypertrophy of anterior and posterior septum; Type III – diffuse hypertrophy sparing only the basal posterior wall; Type IV – apical hypertrophy.[25]

The mechanism of LVOT obstruction (LVOTO) is complex, involving ventricular hypertrophy and abnormalities of the MV apparatus.[26-28] The common pathway is represented by a narrower ventricular outflow cavity with or without distortion of MV leaflet coaptation and subsequent MR. Scenarios more likely to result in LVOTO include asymmetric hypertrophy, a prominent basal septum, a narrowed outflow cavity (< 20-25 mm) , and structural abnormalities of the MV apparatus. The latter includes anteriorly positioned papillary muscles,[29] abnormal insertion of the papillary muscles into the mitral leaflet,[30] elongated mitral leaflets,[31,32] and disturbances in MV annular function (e.g. posterior annular calcification). Echocardiographic delineation of the mechanism of LVOTO is important for planning therapeutic interventions especially in regards to the requirement for, and timing of surgery.

Echocardiographic Examination

In patients with HCM/HOCM, systolic function is usually preserved (LVEF ≥ 55%) or hyperdynamic (LVEF ≥ 65%) and the ventricular dimensions are normal or reduced in size. A smaller percentage (< 5-10%) of patients may have reductions in systolic function with or without chamber dilation, suggestive of late- or end-stage cardiomyopathy. Focal and global systolic dysfunctions are due to disarray of the myocardial fibers with or without fibrosis. In addition, decreases in myocardial performance may be due to atheromatous CAD, hypertrophic involvement of the coronary arteries, reduction of coronary perfusion in a hypertrophied ventricle, or decreased coronary artery vasodilatory reserve.[33]

Wall thickness ranges from normal (≤ 11mm) to greater than 30 mm with varying patterns from apex to base, concentric or asymmetric. The absence of hypertrophy does not rule out HCM/HOCM since development of myocardial thickening may be delayed until the second or third decade, emphasizing the need for routine follow-up of patients with a family history of HCM/HCOM.

Diastolic dysfunction, which may associated with ventricular hypertrophy, myocardial disarray, fibrosis, reduced systolic emptying, and/or coronary artery insufficiency occurs in almost all patients. Relief of LVOTO reduces LV pressures and improves coronary blood flow, myocardial oxygen balance, and ventricular filling.[33] Severity of diastolic dysfunction ranges from normal to restrictive filling patterns, the latter reflecting greater degrees of hypertrophy and fibrosis.

Systolic outflow obstruction whether at the level of the LVOT or at the middle portion of the ventricular cavity results in high systolic flow velocities, incomplete systolic emptying, and increased ventricular cavity pressures. The Doppler profile is described as late peaking (> 1.4 m/s) and "dagger-shaped" in appearance reflective of a dynamic process vs. a fixed one. LVOTO can also be demonstrated using M-mode AoV leaflets, which will open normally but close prematurely in mid-systole due to dynamic obstruction of systolic outflow. Two-dimensional echocardiographic assessment may display the narrowing of the LVOT with or without systolic anterior motion (SAM) of the mitral leaflets and chordae. Specific features of the MV and mitral apparatus associated with SAM and LVOTO include redundant or elongated mitral leaflets, lax chordae, abnormally positioned papillary muscles, and mitral annular dysfunction.

Color Doppler analysis of the LVOT and MV demonstrates aliasing of the CFD jet in LVOT at the level of narrowing consistent with high velocity flow, and across the MV indicating significant MR. This 'Y' shaped CFD is consistent with LVOTO/SAM and MR.

The mechanism of SAM/LVOTO and MR is likely to involve some combination of the Venturi effect and abnormalities of the mitral apparatus. These variables may contribute differently from one patient to another. Nevertheless, delineating the mechanisms of SAM/LVOTO/MR with echocardiography provides useful information when considering the best mode of therapy for patients who are symptomatic from outflow tract obstruction and MR.

Restrictive and Infiltrative Cardiomyopathy (RICM)

Etiology and Pathophysiology

Restrictive cardiomyopathy (RCM) is a pathologic process in which diastolic function of one or both ventricles is severely impaired in the absence of a definitive systemic disease. While several diseases are associated with restrictive filling, only Loeffler's hypereosinophilic endocarditis, endomyocardial fibrosis, and idiopathic restrictive cardiomyopathy qualify as primary restrictive cardiomyopathies.[1,34] Secondary causes of RICM include infiltrative diseases such as amyloidosis, sarcoidosis, and storage diseases (e.g., glycogen storage disease, hemochromotosis), certain drugs (anthracyclines, ergotamine, methysergide, serotonin), and a number of miscellaneous causes (transplant rejection, radiation, cancers, toxins). Collectively, these diseases have significant overlap and have been categorized as restrictive/constrictive, infiltrative, congestive or obliterative cardiomyopathies reflecting the range of clinical and morphological presentations. Congestive cardiomyopathy more accurately reflects this group of disorders due to the presence of either pulmonary venous or vena caval congestion, which results in signs of left and right heart failure, respectively. However, since this term is vague and could include all cardiomyopathies, the diseases in this section will be referred to as "restrictive/infiltrative cardiomyopathies": RICM).

RICM should be suspected when a patient presents with CHF, non-dilated ventricles, dilated atria, diastolic dysfunction, and poor response to medical therapy. While chest X-ray reveals cardiomegaly and a thickened heart, ECG demonstrates low QRS voltage consistent with replacement of the normal myocardium with non- or poorly conducting tissue. Tissue biopsy frequently confirms the diagnosis, however it may also

show non-specific and non-diagnostic fibrotic changes of the endomyocardium and myocardium.[35]

2-D Echocardiographic Evaluation

Establishing the diagnosis and determining the severity of ventricular dysfunction is important to develop establish a treatment plan and prognosis. Differentiation from more treatable causes of heart dysfunction (e.g. CAD, valvular heart disease, pericardial disease) is necessary to allow prompt performance of therapeutic procedures (e.g. pericardiectomy for pericarditis) when indicated.

Amyloidosis is the most commonly reported etiology of RICM and is caused by an abnormal layering of protein within the myocardial tissues including all cardiac chambers, the coronary arteries, cardiac conduction system, and heart valves.[36] The heart has been described 'rubbery' and non-compliant. Although all patients have bi-atrial enlargement, only 20% had ventricular dilation, which was associated with other pathologies (e.g. CAD, primary pulmonary disease). Focal wall motion abnormalities suggest either amyloid or athermatous involvement of the coronary arteries.
A number of echocardiographic features differentiate the amyloid heart from other causes of hypertrophy and/or diastolic dysfunction.[37,38] The echocardiographic appearance of the amyloid heart is classically described as 'speckled', granular, or 'starry skied'. All cardiac tissues may be symmetrically or, infrequently asymmetrically thickened and 'speckled'. Similar to other etiologies of RICM, the atria are enlarged while the ventricular cavity size is often normal or reduced. *However, infiltration of the atrial walls, especially the interatrial septum, differentiates amyloidosis from other causes of CHF.* In addition, RV thickening and speckling are more common with amyloidosis than other diseases. Intracardiac thrombi were found in 26% of patients, with a greater incidence in the atria.

Mortality for patients with cardiac amyloidosis is high, and survival beyond 2-3 years for patients presenting with CHF is less than 50%.[39] Prognosis in patients with amyloidosis is based on myocardial wall thickness, severity of diastolic dysfunction, and systolic function.[34,39] For patients with wall thickness $\leq$ 12 mm the median survival is about 2.5 years, while survival is reduced for those with wall thickness between 12 and 15 mm (1.3 years), and least for those with wall thickness $\geq$ 15 mm (0.4 years). The incidence of systolic dysfunction is 0%, 35%, and 70% in these 3 groups respectively. Prognosis is also related to the severity of diastolic dysfunction, which in turn is correlated with ventricular wall thickening. For patients with milder cardiac involvement, wall thickness is < 15 mm, and Doppler profiles suggest a pattern consistent with abnormal relaxation. In contrast, patients with wall thickness $\geq$ 15 mm have flow profiles suggestive of a restrictive filling defect. Normal RV free wall thickness is < 7 mm. Patients with RV wall thickness $\geq$ 7 mm the right sided Doppler patterns reveal a restrictive filling defect.

Idiopathic restrictive cardiomyopathy is an autosomal dominant disease associated with heart block and skeletal myopathy, which presents in the third and fourth decades.[34,39] Histologic examination reveals variable degrees of interstitial fibrosis throughout the heart. For children under 10 years of age, the survival is less than 2 years, while more than 60% of adults survive beyond 4 years. Echocardiographic evaluation

reveals diastolic dysfunction, relatively normal systolic function, variable myocardial thickening, atrial enlargement with or without thrombi, and pulmonary hypertension.

Endocardial fibroelastosis is found in children and characterized by a thick endocardium.[40] Histology demonstrates infiltration of the endocardium with collagen and elastic tissue causing LV endocardial thickening with or without MV involvement. While the primary form is not associated with other congenital cardiac abnormalities, a secondary form may be found with LVOTO, aortic coarctation, coronary artery abnormalities, or hypoplastic left heart.

Hypereosinophilic syndrome (*Loffler's endocarditis*) and *endomyocardial fibrosis* are a continuum of the same disease differing only by their presenting pathology.[41] Although these diseases are uncommon, their occurrence is greater in parts of Africa and Asia, accounting for as much as 25% of cardiac deaths. While both are the result of cardiac eosinophilia, Loffler's endocarditis presents with significant cardiac hypereosinophilia, while endomyocardial fibrosis represents a later stage characterized mainly by fibrosis. As both diseases progress, endocardial thickening and fibrosis develop with greater involvement of the MV and TV subvalvular apparatus producing valve insufficiency and/or stenosis. Involvement of the ventricular apex results in obliteration of the cavity, which may be further complicated by thrombus formation. Echocardiography demonstrates bi-atrial enlargement, endocardial thickening or deposits along the MV and TV associated papillary muscles, and along the apices of both ventricles. The involved tissues appear bright and echodense consistent with calcium deposition. While impairment to ventricular filling is present, systolic motion of the ventricular walls is usually preserved.

Hemochromatosis and *Sarcoidosis* are infiltrative processes due to iron deposition and non-caseating granulomas respectively. For both diseases, cardiac involvement is rarely seen in the absence of non-cardiac organ involvement. Cardiac involvement is seen in as many as 20-40% of patients.[42,43] In sharp contrast to primary RCMs, systolic dysfunction is typical with varying degrees of diastolic dysfunction. A restrictive filling defect is rare.

A number of miscellaneous etiologies of RICM have also been described including carcinoid, radiation therapy, and various metabolic storage diseases.

Doppler Assessment of Diastolic Function: Restrictive Physiology

The Doppler flow profiles seen with mild diastolic dysfunction are primarily the result of reduced ventricular relaxation while atrial pressures and function are relatively normal. As diastolic function deteriorates, ventricular stiffness increases along with ventricular and atrial pressures. Atrial contractility and compliance subsequently decrease with further reductions in ventricular compliance. Restrictive filling patterns are consistently associated with poor exercise tolerance, CHF, and mortality across a number of different populations including patients with RICM.[37,43-45]

Flow profiles suggesting a restrictive filling include an E/A ratio > 2.0, DT < 150 msec, IVRT < 80 msec, PV_D < 30 cm/s, a $PV_{S/D}$ << 1.0, an PV_{AR} > 35 msec, and a PV_{AR} / $TMDF_A$ duration or peak velocity ratio of > 0.6.[77] These filling patterns result from elevated LAP forcing early transmitral flow (decreased IVRT) into a stiff LV (decreased propagation velocity and DT), which subsequently limits transmitral flow during a diminished atrial contraction (i.e., increased E/A ratio). A relatively larger LA

volume is ejected backwards toward the pulmonary veins during atrial contraction, thus producing an increased PV_{AR} / $TMDF_A$ ratio. Incomplete LA emptying during atrial contraction subsequently limits pulmonary venous inflow during ventricular systole, resulting in a decreased PV_S/PV_D. RV failure is associated with similar Doppler flow velocity profiles into the RA and across the tricuspid valve.

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Echocardiographic Evaluation of Pericardial Disease

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Objectives:	1)	Identify subcategories of pericardial disease;
	2)	Apply Echo for quantitative analysis of LV function in patients with pericardial disease.

I. PERICARDIAL ANATOMY

The pericardium is a sac that normally consists of two apposed serous surfaces that surround a closed potential space. The pericardial space normally contains 5 to 30 mL of serous fluid. This potential space is complex in that various reflections of pericardial tissue create sinuses (in this circumstance a sinus is defined as a depression or cavity formed by bending or curving) within the pericardial sac. The *visceral* (continuous with the epicardium) and *parietal* (thin, dense, fibrous structure that blends with the central tendon of the diaphragm inferiorly and is apposed to the pleural surfaces laterally) pericardium are apposed to create the pericardial space. Conjoined areas of both pericardial surfaces meet to close the ends of the pericardial sac in areas termed *pericardial reflections*. The reflections in the posterior region of the pericardium at the atrial level surround the venae cavae and the pulmonary veins. The reflections that surround the venae cavae and pulmonary veins create a dead ended pocket behind the left atrium termed the *oblique sinus* (Figure 1). A useful anatomical landmark to inspect for accumulated fluid within the oblique sinus can be obtained from TEE images of the left atrial appendage (LAA). At an omniplane of approximately 70° at the mid to high esophageal level the LAA appendage appears within the oblique sinus at screen right. An echolucent signal surrounding the LAA in this view represents fluid within the pericardial space. The reflections in the superior region of the pericardial sac surround the great vessels. The pericardial reflection around the great vessels creates the *transverse sinus* posterior to these structures (Figure 1)[1-4].

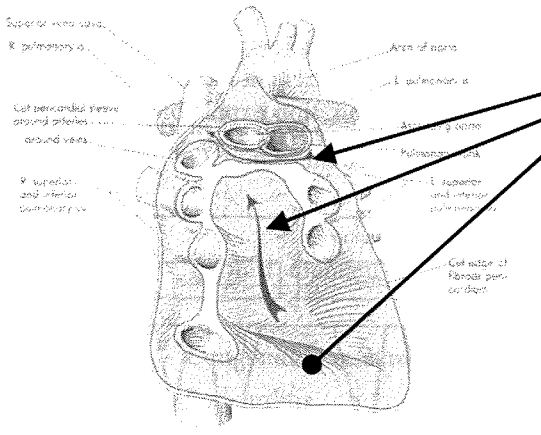


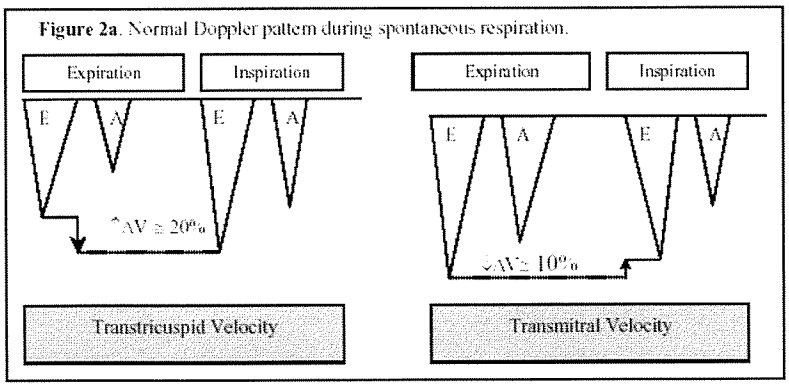
Figure 1. Posterior wall of pericardial sac with heart removed.
Transverse Sinus
Oblique Sinus
Diaphragmatic pericardium

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II. PERICARDIAL PHYSIOLOGY

The definitive function of the pericardium is unclear but it is thought that the normal pericardium provides mechanical restraint to the cardiac chambers and inhibits chamber dilation, especially under conditions of acute volume increase in the heart chambers or within the pericardium. Additionally, the pericardium limits ventricular filling, and may protect against excessive incompetence of atrioventricular valves[5]. Attachments of the pericardium at the diaphragm, at the atrial level and around the great vessels provide restraint to this structure in that expansion beyond these pericardial reflections is inhibited if the heart increases in size; the tough fibrous quality of the parietal pericardium contributes to the pericardium's ability to provide restraint. The pericardium also isolates the heart from the surrounding mediastinal structures (e.g., the lungs and pleural spaces). The pericardium may also provide a lubricating function that facilitates normal myocardial rotation and translation during each cardiac cycle in that the mesothelial layers of the pericardium produce a serous fluid that serves as a lubricant. The pericardium is known to secrete biochemical substances (e.g., prostacyclin) that may play a role in sympathetic neural regulation, coronary vascular tone and cardiac contractility. The pericardium also has a fibrinolytic function in that it contributes to maintaining accumulated blood in a liquid state[1-2,4]. Much of the microphysiologic function of the pericardium is not completely understood.

Normally during spontaneous respiration, changes in intrathoracic pressures are nearly equally transmitted to the pericardial space and intracardiac chambers. The pericardial pressure commonly approximates pleural pressure and will vary with the respiratory cycle demonstrating pressures of approximately –6 mm Hg at end inspiration and –3 mm Hg at end expiration (NB – as measured by a fluid filled, non-balloon tipped catheter). It is the lowering of intrathoracic and pericardial pressure during inspiration that permits increased filling of the right-sided chambers in that venous return is augmented. The true filling pressure of the heart is determined by transmural pressure which is calculated by subtracting the pericardial pressure from the intracardiac pressure. For example, in a spontaneously breathing patient with a right atrial (RA) pressure of 6 mmHg and a pericardial pressure of – 6 mmHg the actual filling pressure is 6 – (-6) = 12 mmHg, during inspiration. The same inspiratory negative intrapleural pressures create a pooling of the increased right ventricular (RV) output in the pulmonary circulation which reduces left atrial (LA) pulmonary venous return. The decrease in LA return results in a decrease in left ventricular (LV) stroke volume and output. Additionally, the decrease in intrathoracic pressure during spontaneous inspiration relative to higher extrathoracic systemic arterial pressures may contribute to a decrease in left heart output[4,6]. Measurable Doppler changes in the transatrioventricular valvular velocities accompany these intrapericardial pressure dynamics. During the inspiratory phase of spontaneous respiration normal transtricuspid velocities increase by approximately 20%. Transmitral velocities normally decrease by approximately 10% during spontaneous inspiration (Figure 2a)[1]. Intermittent positive pressure ventilation (IPPV) will produce opposite changes in velocities (Figure 2b)[7]. The increase in intrathoracic pressures relative to extrathoracic systemic pressures during positive pressure inspiration favors an increase in left heart output. Additionally, the increased intrathoracic pressures during positive pressure inspiration expels blood from the pulmonary veins into the LA and thus augments left heart filling and output[4]. The changes of transatrioventricular valvular velocities have been termed *respirophasic variation.*



The absolute values of these velocities are affected by several physiologic variables that include age, heart rate, rhythm, preload, volume flow rate, ventricular systolic function and atrial contractile function. The transmission of intrathoracic pressures to the intrapericardial structures appears blunted in patients with certain pericardial pathologies (e.g., pericarditis or pericardial effusions severe enough to elicit tamponade physiology[7]).

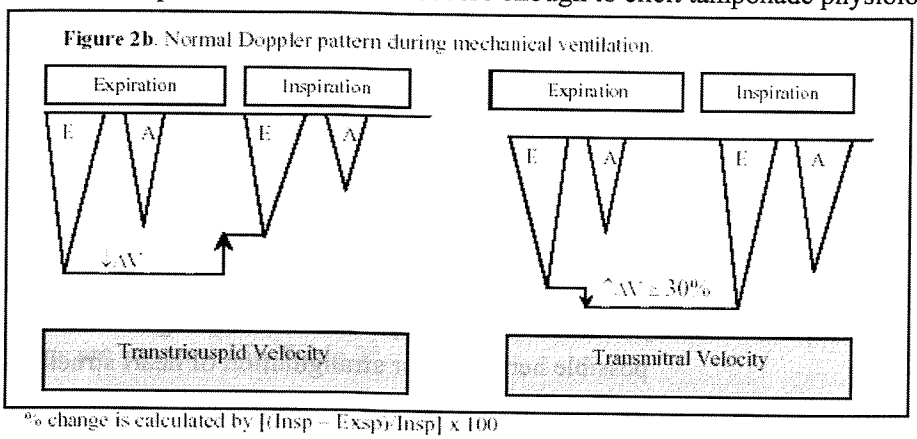


Figure 2b. Normal Doppler pattern during mechanical ventilation.

% change is calculated by [(Insp – Exsp)/Insp] x 100

Note that under conditions of intravascular volume depletion that an alternative profile of transmitral velocity has been described in an animal model. In one report using a canine model 45% of the observed transmitral profiles revealed a slight decrease in maximal amplitude during positive pressure inspiration that even further decreased during expiration and eventually reached a nadir during expiration (Figure 2c). The author's attributed the observation of this pattern to intravascular volume depletion which was manifest as a direct result of reduced pulmonary venous vascular reserve[7].

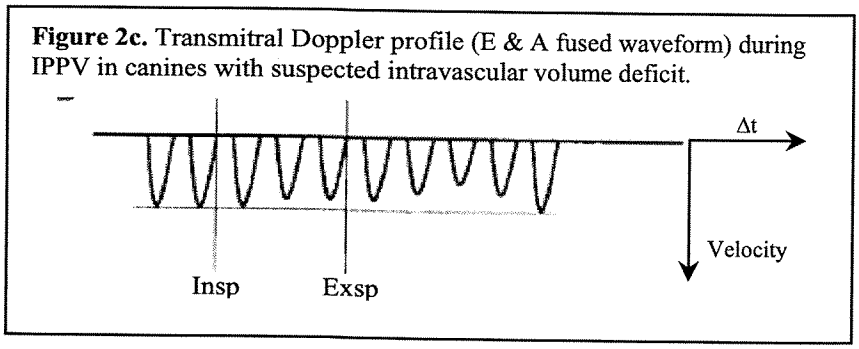


Figure 2c. Transmitral Doppler profile (E & A fused waveform) during IPPV in canines with suspected intravascular volume deficit.

III. PERICARDIAL PATHOLOGY

Echocardiographic evaluation of pericardial disease is performed to rule out specific pathologies that can affect myocardial function. There are five basic categories of pericardial pathology that one must consider (Table 1).

Table 1. General Categories of Pericardial Pathology

Congenital Pericardial Defects
Pericarditis
Pericardial Effusion
Pericardial Tamponade
Pericardial Masses

Congenital pericardial defects are rare and consist of partial or total absence of the pericardium. The significance of pericardial absence varies although appears minor in that most of these patients are clinically asymptomatic (Table 2). One small observational study (n=10) reviewing this disorder found that paroxysmal stabbing chest pain that mimicked coronary ischemia was the most common clinical presentation[8]. Individuals with congenital absence of the pericardium are at increased risk for additional congenital abnormalities in that 30% of these patients will have some additional congenital pathology[3,23].

Table 2. Pathologic Characteristics of Forms of Pericardial Absence

Form	Incidence	Clinical Significance
Total Bilateral Absence	Rare	Mostly asymptomatic
Partial Left Absence	70%	Increased risk for aortic dissection if augmented heart mobility; possible herniation or strangulation of heart structures through the defect with chest pain, shortness of breath, syncope
Partial Right Absence	17%	Increased risk for aortic dissection if augmented mobility of the heart exists as a result of the defect
Overall	0.01%	

Incidence values represent a percentage of the overall incidence of 0.01%.

Echocardiographically, one may observe paradoxic ventricular septal motion, an exaggerated posterior left ventricular wall and possibly the appearance of a dilated right ventricle when assessed through the transthoracic parasternal window. A small series of patients (n=8) from a Japanese group reported more marked alterations in venous return in the right heart (measured in the superior vena cava) than in the left heart (measured in the pulmonary veins)[9]. CXR and cardiac MRI are the two imaging modalities that appear to be most useful in making the definitive diagnosis of congenital absence of the pericardium[9].

Pericarditis is an inflammation of the pericardium and can occur as the result of multiple pathologies. Pericarditis can also present as either a *chronic* or *acute* process. Clinically, one examines the patient suspected of having pericarditis for the presence of the classic triad of chest pain, ECG evidence of pericarditis (e.g., diffuse ST segment elevations), and a pericardial rub on auscultation. Etiologies of pericarditis include the following:

- Infections (e.g., postviral, bacterial, especially tuberculosis)
- Malignant (e.g., metastatic disease or direct extension of a pulmonic or breast tumor)
- Inflammatory (e.g., post-myocardial infarction (Dressler's syndrome), uremia, collagen vascular disease, postcardiac surgery)
- Intracardiac-pericardial communications (e.g., chest trauma, post-interventional catheter procedures, postmyocardial ventricular rupture)[2].

Echocardiographically, one inspects the pericardium for evidence of thickening that appears as an increase in the brightness, or echogenicity of the ultrasound signal. M-mode analysis of a patient with pericarditis will reveal multiple parallel ultrasound reflections and can be helpful to discern the thickened pericardium (Figure 3). Multiple echo windows should be obtained to verify the diffuse or localized nature of the pericarditis. A pericardial effusion may be present concurrently with pericarditis[2]. Echocardiography lacks sufficient fidelity to quantify the degree of pericarditis and thus carries an unreliable sensitivity and specificity for detecting this disorder compared to other imaging techniques (e.g, cardiac MRI).

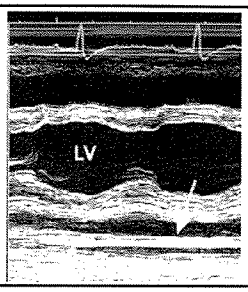


Figure 3. M-mode image (the white arrow highlights a thickened pericardium).

Otto.Textbook of Clinical Echocardiography 3ed. 2004

Pericardial effusion is a collection of fluid within the pericardial sac that can either be diffusely distributed within the pericardium or localized. Depending on patient position and the amount of volume contained in the effusion different patterns of fluid distribution can be seen assuming that the pericardial space does not contain fibrous adhesions, or a mass that may redirect accumulating fluid. Echocardiographically, a pericardial effusion appears as an echolucent signal immediately adjacent to the epicardium. General guidelines on pericardial effusion size and fluid distribution patterns are presented in Table 3[2].

Table 3. Pericardial Effusion Charaharacteristics

Severity	Width by Echo	Volume (mL)	Localization Region
Small	< 10 mm	< 100	Behind posterior LV wall
Moderate	10-15 mm	100-150	Expand laterally and apically
Large	> 15 mm	> 500	Evenly distributed around the heart

The clinical significance of a pericardial effusion relates to the amount of fluid present and the time course over which it accumulates. Large, chronic effusions are frequently associated with excessive antero-posterior heart motion as well as counterclockwise rotation in the horizontal plane. Effusions can lead to cardiac translation within the pericardial space that can create the ECG finding termed *electrical alternans* (Figure 4)[1]. Electrical alternans is the regular occurrence of alternating heights of the QRS complex that may also involve the P, T, U waves and ST segments; this finding is not specific to pericardial effusions[10].

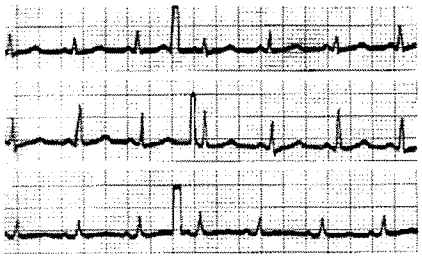


Figure 4. Electrical alternans on the ECG of a patient with a pericardial effusion.

Goldschlager & Goldman. Principles of Clinical Electrocardiography. 13th ed. 1989.

Pericardial effusions are highly common in cardiac surgical patients (85%) and generally peak by the 10th postoperative day[1]. The relevance of a pericardial effusion relates to the volume of fluid that accumulates and the chronicity with which it accumulates (Figure 5a)[2]. Over time the pressure in the pericardial space will increase as the effusion grows in volume and eventually intrapericardial pressure will rise above the cardiac chamber pressures. When intrapericardial pressure increases to the point that cardiac chamber filling is compromised the patient is considered to have developed *tamponade physiology* resulting in the clinical finding of **pericardial tamponade**. Additionally, when the amount of accumulating fluid causes a more extreme increase in pericardial pressure (i.e. the pressure-volume relationship has reached the steep point on the curve which relates these two physiologic variables (Figure 5b)) *ventricular interdependence* will develop[5,11]. Note the adaptive nature of the pericardium that is observed with slowly accumulating effusions in Figure 5b. With ventricular interdependence as the right ventricle fills the interventricular septum will shift towards the left and thus compromises left ventricular filling and left ventricular output as evidenced by a decrease in systemic blood pressure (Figure 5c)[12]. Loss of the y-descent in the CVP tracing is also characteristic of tamponade.

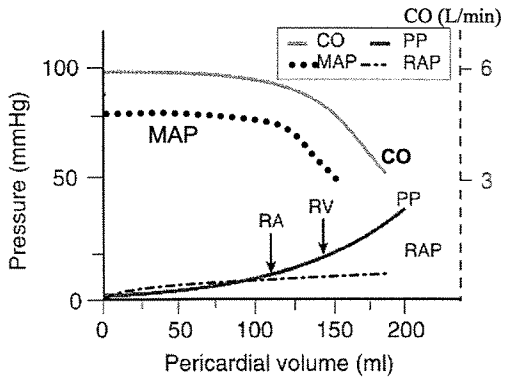
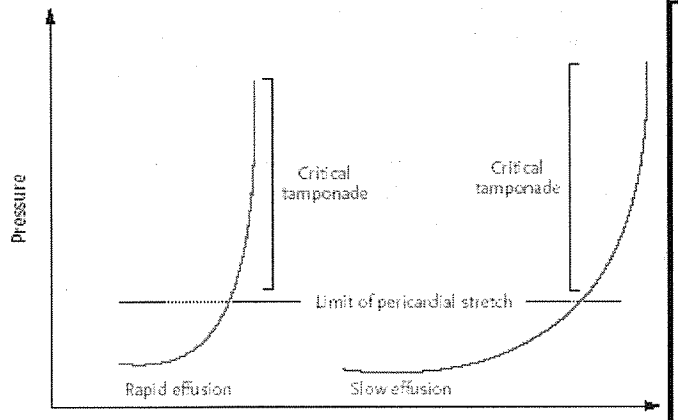


Figure 5a . Graphed relationship of pericardial pressure (PP), RA pressure (RAP), mean arterial pressure (MAP) and cardiac output (CO). When PP exceeds RAP (designated by the point marked RA) the MAP & CO begin to fall. When RV pressure is exceeded by PP the MAP & CO fall further.

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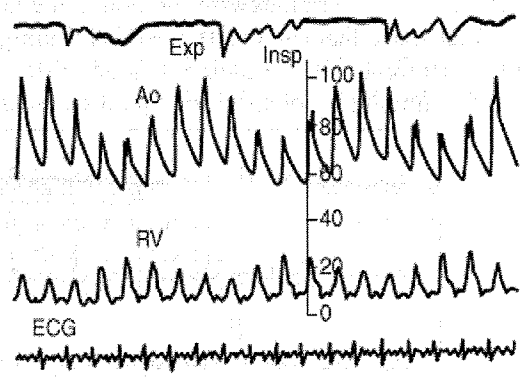
Tamponade physiology occurs when the pericardial pressure exceeds cardiac chamber pressure; lower pressure chambers (e.g., RA in systole) will be affected initially followed eventually by all cardiac chambers if pericardial pressures continue to rise. In fully developed tamponade all cardiac chambers will have elevated and equal diastolic pressures. Loculated effusions can elicit tamponade physiology with a considerably smaller volume of fluid than unloculated effusions if the fluid collection is strategically located near a low-pressure cardiac chamber (e.g., the right or left atrium). Echocardiographic examination must involve a 2D interrogation from multiple windows to assess for the potential presence of one or more loculated effusions.

Figure 5b. Pericardial Pressure to Volume Relationship



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Figure 5c. Tamponade Hemodynamics



Braunwald 2005.7th ed.

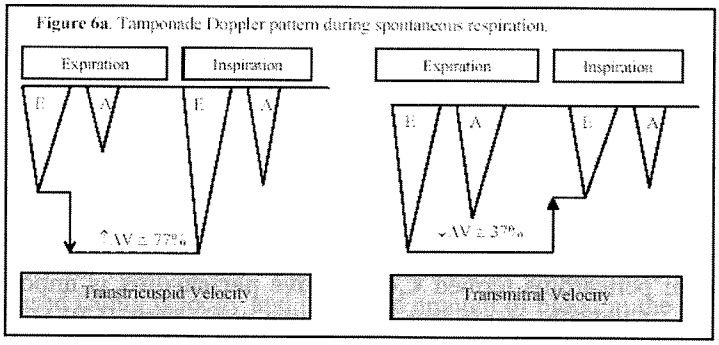
Pericardial effusions can create a host of echocardiographic findings that include:

- An echolucent signal of variable width (Table 3) adjacent to the pericardium (note that the echolucent signal can brighten if the pericardial space is filled with blood that has clotted as may be seen with *hemopericardium*)
- Fibrinous stranding within the pericardial fluid may be seen in patients with long-standing pericardial disease
- Regional septal wall motion abnormalities
- MV & TV prolapse
- Systolic anterior motion (SAM) of the anterior mitral leaflet
- Early systolic closure of the aortic valve
- Mid-systolic notching (partial closure) of either the aortic valve or pulmonic valve

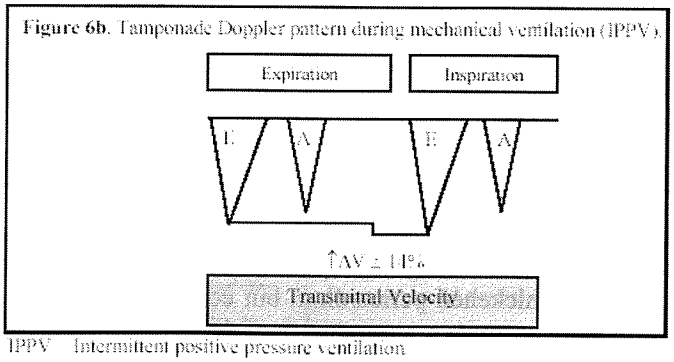
When pericardial effusions grow to an extent that elicits pericardial tamponade there are a host of echocardiographic findings that can be observed[2]:

- RA systolic collapse, or inversion (inversion for > 1/3 of systole has a 94% sensitivity and 100% specificity for tamponade)
- RV diastolic collapse (slightly less sensitive and specific for the diagnosis of tamponade)
- Reciprocal respiratory changes in RV and LV volumes (during spontaneous inspiration an increase in RV volume will occur that will result in a shift of the interventricular septum towards the left ventricle in diastole; these changes reverse during expiration-*pulsus paradoxus* will be observed on the arterial waveform under these physiologic conditions)
- Inferior vena cava (IVC) plethora (dilated IVC with < 50% inspiratory reduction in diameter near the IVC-RA junction; this a sensitive (97%) but nonspecific indicator of tamponade)

Note that Doppler evaluation of tamponade physiology can also provide useful information to confirm the diagnosis of pericardial tamponade. As seen during spontaneous respiration in normal trans-atrioventricular valve Doppler profiles there is a decrease in transmitral velocity flow profiles during inspiration (Figure 2a). In severe tamponade this decrease can be as much as 37% (Figure 6a). The transtricuspid valve velocities increase by as much as 77% with tamponade physiology during spontaneous respiration[13]. The magnitude of the transatrioventricular velocities will be reduced in absolute value although the respirophasic variation will be accentuated in pericardial tamponade during spontaneous respiration. The mechanism accounting for the exaggerated respirophasic variation in tamponade is an insulation of the heart from the intrathoracic pressure changes associated with the respiratory cycle.

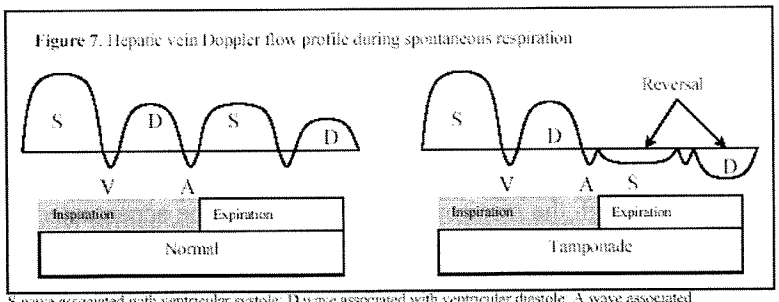


Doppler interrogation of transmitral valve velocities during intermittent positive pressure ventilation will reveal a markedly different pattern than seen with spontaneous respiration. According to work done in a canine model the normally observed dynamics in transmitral valvular velocities seen in pericardial tamponade will be opposite in direction and markedly attenuated (Figure 6b)[7]. Thus the characteristic respirophasic variation in transatrioventricular velocities seen with tamponade in spontaneously breathing patients is absent during positive pressure ventilation. However, consistent with the pattern seen in spontaneously breathing patients with tamponade there will be an overall decrease in the amplitude of the flow velocities during mechanical ventilation compared to values obtained in individuals without tamponade[5,7].



IPPV Intermittent positive pressure ventilation

In addition to interrogation of the trans-atrioventricular valve velocities, assessment of the *hepatic vein flow profiles* can also provide useful diagnostic information in spontaneously breathing patients suspected of having tamponade physiology. Normally the pulse wave Doppler profile of the hepatic vein is bimodal with the forward flow during systole being equal to or greater than the forward flow during diastole throughout the respiratory cycle. The systolic and diastolic forward velocities increase during inspiration with spontaneous respiration. Under conditions of tamponade physiology the systolic and diastolic forward flow velocities also increase in magnitude during inspiration but during expiration there is a marked reduction, or reversal of both the S and D waves which is not seen in normal subjects (Figure 6)[13]. Data on respirophasic changes in the hepatic veins during positive pressure ventilation is not available and thus evaluation of the hepatic vein profiles with TEE during positive pressure ventilation is not useful at present.



S wave associated with ventricular systole; D wave associated with ventricular diastole; A wave associated with atrial contraction; V wave associated with recoil of the tricuspid annulus following ventricular systole

A final Doppler echocardiographic quantitative method that can be integrated into the assessment of a pericardial effusion and tamponade physiology is measurement of the isovolemic relaxation time (IVRT). In one transthoracic study that compared spontaneously breathing normal adults to individuals with tamponade as well as to individuals with an effusion, but no tamponade, a significantly prolonged IVRT was observed in patients with tamponade compared to normal individuals. Normally the IVRT is < 100 msec in most age groups (mean value of 82 msec was observed in this study) and a mean value of 104 msec was observed in the tamponade group that was studied ($P < 0.01$)[13].

The evaluation of a subtype of pericarditis termed **Constrictive pericarditis** is important to be aware of when diagnosing pericardial disease. Clinically significant constrictive pericarditis (CP) will affect ventricular filling. CP has been reported to occur in 0.2 – 0.3% of cardiac surgical patients and has a number of pathophysiologic features that include: fibrotic pericardium, inflamed pericardium, calcific thickening of the pericardium, abnormal diastolic filling, a narrow RV pulse pressure (i.e. the systolic RV pressure is normal but the diastolic RV pressure is elevated), a prominent early diastolic RV pressure dip and later plateau (termed the "square root sign" (Figure 8)[1,14]. Additionally, the RA pressure exhibits a pronounced systolic drop (y descent followed by an increase and then plateau) that appears as an "M-like" wave on the CVP tracing (Figure 8).

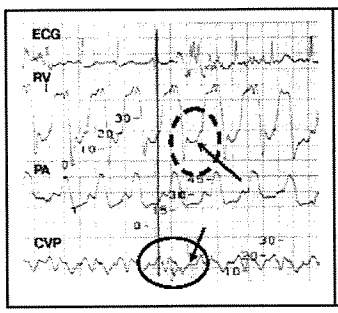


Figure 8 Hemodynamic tracings from an intraoperative patient with constrictive pericarditis on intermittent positive pressure ventilation. The "square root sign" is indicated within the broken circle (note the elevated diastolic plateau indicated by the broken arrow). The "M-like" CVP waveform with the post-systolic plateau (solid black arrow) is indicated within the solid circle.

The diagnosis of CP is made by using a combination of 2D echocardiographic analysis and spectral Doppler analysis. The 2D echocardiographic exam will generally reveal a thickened, highly reflective pericardium in patients with CP. TEE can detect the thickened pericardium but computed tomography and magnetic resonance imaging are considered to provide more accurate measurements of the pericardial thickness. Table 4 lists a number of nonspecific echocardiographic findings associated with CP[1].

Table 4. 2D-Echocardiographic Findings Associated with CP

Paradoxical ventricular septal motion
Ventricular septal "bounce"
Diastolic flattening of the posterior LV
Premature mid-diastolic pulmonary valve opening
Spontaneous inspiratory leftward shift of the atrial & ventricular septum
Enlarged hepatic veins
Dilated IVC without variation in size during respiration
Normal ventricular size
Normal or enlarged atria with reduced wall excursion

Doppler assessment of the transmitral blood flow has proven to be a useful diagnostic and prognostic tool for individuals with CP. By color Doppler assessment patients with CP may exhibit tricuspid or mitral valve incompetence. Similar to pericardial tamponade, the spectral Doppler profile of most patients with CP will demonstrate an exaggerated respirophasic variation of the transmitral velocity (i.e. it will decrease by approximately 25% during spontaneous inspiration). Note that up to 20% of patients with CP will not exhibit this Doppler finding although the application of preload reducing maneuvers (e.g., reverse Trendelenberg position) may be useful to amplify the transmitral velocity respiratory variation that is expected in these patients[15]. Spectral Doppler assessment of pulmonary vein flow will reveal a systolic to diastolic ratio < 1[16]. The thickened pericardium insulates the intrapericardial structures from the intrathoracic pressure changes associated with the respiratory cycle and produces an exaggerated respirophasic variation as a result (as seen in tamponade); similar respirophasic variation is observed in the pulmonary veins with CP and the S:D ratios observed are similar to those of patients with restrictive myocardial pathology (e.g., the pulmonary vein diastolic flow velocity will be greater than the systolic flow velocity in patients with a restrictive LV filling pattern, or S:D ratio < 1)[16].

During mechanical ventilation the transmitral E-wave velocity will increase with early inspiration in CP[14,17]. The increase in intrathoracic pressure expels blood from the extrapericardial pulmonary veins into the left atrium which results in an increase in transmitral flow velocity. Given that the CP pericarditis limits the volume of both cardiac chambers the inspiratory increase in transmitral E wave velocity will be accompanied by a simultaneous decrease in transtricuspid E wave velocity for two reasons. First, the increase in intrathoracic pressure will limit right sided filling and second, the increased filling velocity and increased amount of blood in the LV will result in a shift of the interventricular septum to the right and thus compromises RV filling during inspiration (i.e. ventricular interdependence is demonstrated). These changes in the transmitral velocities appear to be reversible with surgical treatment (pericardial stripping or pericardiectomy) of CP although this therapy has been associated with LV dilation and transient ventricular diastolic dysfunction[18].

Much has been written about making the distinction between CP and restrictive cardiomyopathy (RCM). Both of these pathologies share the common pathophysiology of decreased left ventricular compliance, but different mechanisms account for the decreased compliance in each case. In CP the decreased compliance relates to the restrictive nature of the thickened pericardium while in RCM the restriction is related to pathology within the myocardium (e.g., infiltrative disease of the muscle or hypertrophy). Recall that decreased LV compliance will present a restrictive LV filling pattern (Figure 9).

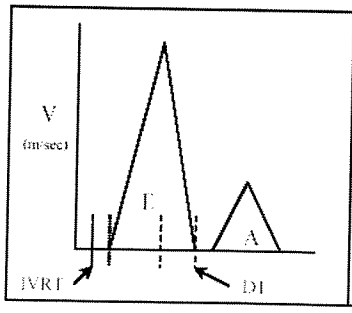


Figure 9. Restrictive LV filling spectral Doppler profile characterized by an E:A ratio of > 2, a reduced deceleration time (DT < 150 msec, and a reduced isovolemic relaxation time (IVRT < 60 msec

Savage and Aronson. Intraoperative Transesophageal Echocardiography. 2005

The differentiation of CP from RCM is best approached by assessing the patient for the described exaggerated respiratory variation in transmitral Doppler flow velocity profiles and for echocardiographic, or cardiac MRI/high resolution CT scan evidence of pericardial thickening. This exaggerated respirophasic variation has been demonstrated in anesthetized patients using TEE[17]. Newer echocardiographic modalities that have been used to differentiate CP from RCM include M-mode propagation velocities (Vp), Doppler tissue imaging (DTI) at the level of the mitral annulus, and Doppler myocardial velocity gradients (MVGs)[19-22].

Pericardial masses include benign pericardial cysts, pericardial neoplasms (mesothelioma, sarcoma, and teratomas are all gratefully rare), and tumors that invade the pericardium from neighboring tissue (e.g., breast or lung). Pericardial cysts are usually round and asymptomatic but can be associated with chest pain, dyspnea, cough, arrhythmias, compression of the pulmonary vein, left atrium or pulmonary vein. Any of these masses may inhibit cardiac chamber filling and result in hemodynamic compromise if they grow large enough in a critical location. Pericardial thrombus formation is another pericardial mass that may precipitate hemodynamic compromise, most commonly in the post-surgical state. Hemopericardium may result from a number of different pathologies (e.g., aortic dissection with extravasation of blood into the transverse sinus, percutaneous catheter interventions, pacer wires or post-infarction myocardial necrosis and rupture).

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Transesophageal Echocardiography During Left Ventricular Assist Device Surgery.

Lecture Goals:

1. Describe the essential components of a TEE exam in these patients
2. Describe how TEE can assist in perioperative anesthetic management of LVAD patients
3. Provide case examples of perioperative TEE in LVAD patients
4. Review the relevant content for the PTEeXAM (content outline for the PTEeXAM)

Surgical treatment of heart failure has been shown to be effective and appropriate in certain settings. For example, end stage heart failure patients (New York Heart Class IV) have a one-year mortality of 75% which decreases to 15% following cardiac transplantation (6), (7). Unfortunately 16,000 patients could benefit from transplantation and less than 3,000 organs are available each year (7). Ventricular assist device (VAD) therapy may provide an option for those patients who are unable to receive transplants due to a lack of organ availability or because they are not candidates for cardiac transplantation. Ventricular assist devices are currently placed for 3 indications: 1. for temporary support as a bridge to transplantation, 2. for temporary support until the heart recovers, 3 for long-term treatment of patients who are not transplant candidates, and who are not expected to recover ventricular function (destination therapy). When compared with medical therapy, left ventricular assist devices have been shown to double one year survival and triple two year survival (7) (8).

Patients with end-stage heart failure, who are not transplant candidates, have been found to have increased survival and improved quality of life following placement of left ventricular assist devices (LVADS) (1). With the limited availability of organs and with the large number of patients who are not candidates for cardiac transplantation, LVAD therapy will become more common. Increasingly these patients will present for cardiac and noncardiac surgery. Perioperative TEE provides real time diagnostic and monitoring information that is essential in managing these patients during surgery and in the ICU.

This lecture will describe the perioperative use of TEE in patients presenting for LVAD placement and will focus on the common echocardiographic features and anesthetic concerns involved in all LVAD cases. A detailed discussion of the individual characteristics of different left ventricular assist devices will not be addressed and the reader is encouraged to see references at the end of this synopsis. Although it will not be the focus of this lecture, it is of paramount importance that a discussion of the perioperative plan occurs prior to induction of anesthesia. Things which should be discussed include: the patient's current condition, the specific idiosyncrasies of the device to be inserted, and the planned insertion sites of the inflow and outflow cannulae. As with all cardiac surgical procedures, good communication between anesthesiologists, surgeons, perfusionists, and device representatives helps improve patient care.

In every case of LVAD placement the following *essential components of a TEE exam* must be performed:

Prebypass:

1. Aortic valve exam; significant AI? (if present AV must be replaced or oversewn)
2. Intracardiac shunts (interatrial communications will result in shunting→ hypoxia)
3. RV function (Tricuspid regurgitation, lateral tricuspid annular plane systolic excursion)
4. Intracardiac Thrombus (left atrial appendage, left ventricular apex)
5. Aortic atherosclerosis (epiaortic scan of outflow insertion site)

Intraoperative (while on cardiopulmonary bypass):

1. Assist surgical positioning of cannulae (LV apex, Trans-septal, Trans-AV)

Weaning from Bypass:

1. LV inflow cannulae located at the apex must be directed away from the septum
2. Adequate flow (appropriate LV end diastolic dimension, adequate mean arterial pressure)
3. Inspection of interatrial septum (IAS) to ensure absolutely no patent foramen ovale (PFO)
4. Inspection of aortic valve [evaluation for aortic insufficiency (AI)]
5. Right ventricular function (need for nitric oxide, phosphodiesterase inhibitors, β-agonists, levosimendan) (right coronary air embolus)
6. De-airing

Post Bypass:

1. Right ventricular function
2. Doppler of inflow cannula (look for septal occlusion) (CWD < 230 cm/sec)
3. Intact interatrial septum (no patent foramen ovale)
4. Volume status

Post OP (ICU):

1. Hypoxia → look for patent foramen ovale, right ventricular failure
2. CVA → look for intracavitary thrombus, patent foramen ovale, air
3. Hemodynamic instability→ look for hypovolemia (bleeding), Tamponade, Right ventricular failure.

New Advances / technology:

Although successful weaning from left ventricular assist devices is difficult (2) (10) new technology may assist in identifying candidates for weaning and explantation. Pressure area loops may be generated using automated border detection (TG SAX view) and high-fidelity intraventicular pressure recording (4). In addition tissue Doppler imaging may predict myocardial recovery during circulatory support with left ventricular assist devices (3). Also, three dimensional echocardiography may improve accuracy of volume measurements in these patients (8), and recent evidence from a small study suggests that medical treatment combined with LVAD therapy may help reverse the remodeling process and enable successful weaning and explantation (10).

Summary:

With the limited availability of organs and with the large number of patients who are not candidates for cardiac transplantation, LVAD therapy will become more common. Increasingly, these patients will present for cardiac and noncardiac surgery. Perioperative TEE provides real time diagnostic and monitoring information that is essential in managing these patients during surgery and in the ICU. As these devices become more prevalent, it is imperative that the perioperative echocardiographer become familiar with these patients, and their unique concerns.

Practice Questions:

1. Which of the following is an essential component of the prebypass exam in patient's undergoing LVAD placement?
   A. Evaluation of RV function
   B. Evaluation for septal defects
   C. Evaluation for intracadiac thrombi
   D. Evaluation for aortic insufficiency
   E. All of the above

2. A high (>4 m/sec) continuous wave Doppler LVAD inflow velocity most likely indicates which of the following?
   A. Thrombus obstructing device outflow
   B. Device malfunction
   C. Septal obstruction of device inflow
   D. Hypovolemia
   E. Right ventricular failure

3. Which of the following would be expected to be the next cardiac surgery an LVAD patient requires?
   A. Tricuspid valve repair
   B. Myomectomy for septal occlusion of LVAD inflow
   C. Cardiac transplantation
   D. Mitral valve repair
   E. Pulmonary thromboendarterectomy

4. Which of the following Prebypass valvular abnormalities is most concerning in a patient undergoing LVAD placement?
   A. Tricuspid insufficiency
   B. Pulmonic insufficiency
   C. Aortic insufficiency
   D. Mitral insufficiency
   E. All of the above are concerning

5. Which of the following is an indication for LVAD placement?

A. 25 year old with post partum dilated cardiomyopathy awaiting transplantation
B. 69 year old with ischemic cardiomyopathy who is not a transplant candidate due to elevated pulmonary vascular resistance.
C. 17 year old with idiopathic dilated cardiomyopathy in whom it is hoped the myocardium will recover.
D. 3 year old with post viral cardiomyopathy
E. All of the above are candidates for LVAD therapy

6. Which of the following best describes the diastolic function that would be expected in a patient undergoing LVAD implantation?
   A. normal diastolic function
   B. Grade 1 (impaired relaxation)
   C. Grade 2 (pseudonormal diastolic dysfunction)
   D. Grade 3 (reversible restrictive diastolic dysfunction)
   E. Grade 4 (irreversible restrictive diastolic dysfunction)

7. Which of the echocardiographic findings would be consistent with a patient undergoing LVAD implantation?
   A. Lateral mitral annular tissue Doppler E_M = 8.6 cm/sec
   B. Ratio of $E/E_M > 15$
   C. Transmitral Color M-mode propagation flow velocity (VP) = 53 cm/sec
   D. Left atrial diameter = 39 mm
   E. Isovolumetric relaxation time =100 msec

8. Which of the echocardiographic findings would be consistent with a patient undergoing LVAD implantation?
   A. LV free wall thickness = 22 mm
   B. Dp/dt = 1600 mmHg/sec
   C. FAC = 50%
   D. FS = 28%
   E. LV end diastolic diameter = 70 mm

Dp/dt = change in pressure with respect to time during isovolumetric left ventricular contraction, FAC = fractional area change, FS = fractional shortening, LV = left ventricle, E = early transmitral inflow velocity as determined using pulse wave Doppler, E_M = early lateral mitral annular tissue Doppler velocity.

Answers:

1. E
2. C
3. C
4. C
5. E
6. E
7. B

8. E

Explanations:

1. *Answer = E,* all of the above are essential components of the prebypass exam. RV function is important, because if the RV doesn't deliver blood to the LV then the device can't pump blood from the LV to the systemic circulation. Optimize ventilation to avoid elevated pulmonary vascular resistance due to hypoxia, acidosis and hypercarbia; add phosphodiesterase inhibitors, beta agonists, and inhaled nitric oxide if needed. A septal defect will result in a right to left shunt causing hypoxemia and potentially a paradoxical embolus. Intracardiac thrombi can also be a source of embolus or device occlusion. AI will decrease device output by causing a recirculation of flow: LV→LVAD→Ao→AV
   References: 1, 7, 9

2. *Answer = C,* Septal obstruction as indicated by a high peak inflow velocity can occur following LVAD insertion. A peak velocity of 230 cm/sec is frequently quoted (9), and this is for a device with a stroke volume of 65 ml and an inflow cannula diameter of 16mm. Obviously the specific hemodynamics will depend upon the device, but in general a high peak inflow velocity on CW Doppler or turbulent inflow on color flow Doppler is concerning for obstruction. A thrombus obstructing the ***Inflow*** (NOT the outflow) cannula could cause elevated inflow velocities. References: 1, 7, 9

3. *Answer = C,* cardiac transplantation is the most likely next surgery. One of the primary indications for LVAD placement is as a bridge to cardiac transplantation.
   References: 1-10

4. *Answer = C,* Aortic insufficiency is a huge problem for a patient undergoing LVAD placement. As explained in answer 1 above, AI decreases LVAD output, lowers systemic perfusion, and causes LV distention. In these patients the valve needs to be replaced or oversewn. If it is hoped that the patient might ultimately be weaned and explanted, (temporary support) the valve should be replaced.
   References: 1, 7, 9

5. *Answer = E,* All of the above are candidates for LVAD therapy
   References: 1-10

6. *Answer = E,* Grade 4 (irreversible diastolic dysfunction) is characteristic of end stage heart failure which is likely present in all patients undergoing LVAD therapy.
   Reference: 11

7. *Answer = B,* A Ratio of $E/E_M > 15$ is indicative of high left atrial filling pressures. This would be consistent with end stage heart failure. The other choices listed would be indicative of normal diastolic function and normal left atrial filling pressures.
   Reference: 11

8. *Answer = E,* A LV end diastolic dimension of 70 mm would be consistent with severe LV dilation. A LV free wall thickness of 22 mm is severely hypertrophied; unlikely in a patient with end stage heart failure requiring an LVAD. The other measurements are consistent with normal systolic function which is also inconsistent with end stage failure.
   References: 1, 7, 9

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Transesophageal Echocardiographic Evaluation of Diastolic Function

NOTES

Hemodynamics

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Introduction

Quantitative Doppler assessment of hemodynamics is largely achieved with the use of two principles or equations:

1) Bernoulli's Equation

2) Continuity Equation

Other assessments may be based on patterns of Doppler flows and/or modifications of these two equations.

Bernoulli's Equation: Pressure Gradients

The **Bernoulli** equation relates flow velocity to **pressure gradient** (**PG**) between two points in a tube. Measurement of blood flow velocity (and pressure gradient) is related to the amount of blood flow, the size of the orifice through which blood is flowing, and the compliance on each side of the area assessed.

Bernoulli Equation:

$$\textbf{PG mmHg} = 1/2\rho(V_2^2 - V_1^2) + \rho(dv/dt)dx + R(v)$$

Convection acceleration | Flow acceleration | Viscous friction

$$\textbf{PG mmHg} = 1/2\rho(V_2^2 - V_1^2)$$

PG = pressure gradient across the area of interest

ρ = density of blood; 1.06×10^3 kg/m^3

V_1 = peak velocity of blood flow proximal to area of interest

V_2 = peak velocity of blood flow across the area of interest

In clinical medicine the Bernoulli equation is simplified by ignoring the effects of flow acceleration, viscous friction, and V_1. Thus the PG can be obtained by simply measuring the peak velocity (V_2) of blood flow across the lesion of interest.

Simplified Bernoulli Equation:

PG $= 4V_2^2$

Measurement of Intracavitary Pressures

Intracavitary and pulmonary arterial pressures can be measured by combining a Doppler derived PG across a regurgitant jet and a known or estimated pressure either proximal or distal to the chamber of interest.

Table 1: Equations for cardiopulmonary pressures using the Bernoulli equations and assessment of pressure gradients from time velocity integrals of valvular regurgitation.

Pressure measured	**Equation**
RVSP or PASP	$= 4(V_{TR})^2 + RAP$
MPAP	$= 4(V_{early\ PI})^2 + RAP$
PADP	$= 4(V_{late\ PI})^2 + RAP$
LAP	$= SBP - 4(V_{MR})^2$
LVEDP	$= DBP - 4(V_{AIend})^2$

RVSP = right ventricular systolic pressure; PASP, PADP = pulmonary artery systolic and diastolic pressure; MPAP = mean pulmonary artery pressure; LAP = left atrial pressure; LVEDP = left ventricular end diastolic pressure; V = peak velocity; TR = tricuspid regurgitation; PI = pulmonic valve insufficiency; MR = mitral regurgitation; AI = aortic insufficiency; RAP = right atrial pressure; SBP = systolic blood pressure; DBP = diastolic blood pressure

Right atrial (RAP) or central venous pressure (CVP) is used for the Doppler estimation of other right sided pressures. It can be measured directly with a central venous catheter or estimated with two-dimensional echocardiography by visualizing the inferior (IVC) or superior (SVC) vena cava from esophageal windows. The diameters of the cavae and the change with respiration are measured to estimate right atrial pressure.

Caval Diameter	Change With Respiration	Estimated Right Atrial Pressure
Small (< 1.5 cm)	Decrease > 50%	0-5 mmHg
Normal (1.5-2.5 cm)	Decrease by = 50%	5-10 mmHg
Normal (1.5-2.5 cm)	Decrease by < 50%	10-15 mmHg
Dilated (>2.5 cm)	Decrease by < 50%	15-20 mmHg
Dilated (>2.5 cm)	No Change	> 20 mmHg

Right ventricular systolic pressure (RVSP) and pulmonary artery systolic pressure (PASP)

Using the simplified Bernoulli equation the peak velocity of the tricuspid regurgitant jet is used to calculate the PG between the right ventricle (RV) and right atrium (RA). The peak TR velocity is obtained by placing the continuous wave Doppler beam parallel to the regurgitant jet. By adding a known or estimated RAP or CVP to the RV-RA pressure gradient the RVSP is estimated. For patients without significant pulmonic valve stenosis or right ventricular outflow tract obstruction the RVSP and PASP are similar.

$$\textbf{RVSP or PASP mmHg} = 4V_{TR}^2 + \textbf{RAP mmHg}$$

Pulmonary artery mean (mPAP) and diastolic (PADP) pressures

These pressures are determined from the pulmonic valve regurgitation (PI) flow profile. After placing the continuous wave Doppler beam parallel to the regurgitant jet, the peak early diastolic velocity is obtained to measure the early diastolic gradient between the pulmonary artery and the right ventricle using the simplified Bernoulli equation. This gradient is added to a known or estimated right atrial pressure to yield the mean pulmonary artery pressure (mPAP).

$$\textbf{mPAP mmHg} = 4(V_{early_{PI}})^2 + \textbf{RAP mmHg}$$

$$\textbf{PADP mmHg} = 4(V_{late_{PI}})^2 + \textbf{RAP mmHg}$$

The **Left Atrial Pressure (LAP)** is measured using the mitral regurgitation (MR) flow profile. The peak ventriculo-atrial gradient is calculated from the peak velocity of the mitral regurgitation (MR) flow profile using the simplified Bernoulli equation. The pressure gradient is subtracted from the systemic systolic blood pressure, which is similar to the left ventricular systolic pressure (in the absence of aortic stenosis) to yield the left atrial pressure (LAP).

$$\textbf{LAP mmHg} = \textbf{SBP mmHg} - 4(V_{MR})^2$$

Left ventricular end diastolic pressure (LVEDP) can be assessed using the aortic valve regurgitation (AI) velocity profile. The calculated aortic-ventriculo gradient, measured from the peak end diastolic velocity, is subtracted from the systemic diastolic pressure (DBP) to yield the left ventricular end diastolic pressure.

$$\textbf{LVEDP mmHg} = \textbf{DBP mmHg} - 4(V_{AIend})^2$$

Left atrial and left ventricular pressures can also be estimated from velocity profiles and patterns across the mitral valve and within the pulmonary vein described in greater detail in the section of diastolic function. **(Table 2)**. Flow patterns consistent with severe diastolic dysfunction are found in patients with elevated left sided pressures.

Table 2: Parameters associated with elevation in left heart pressure and/or decrease in diastolic function.

Parameters consistent with LVEDP, LAP, or PCWP > 15-20 mmHg	
E/A	> 1.5
IVRT	< 80msec
DT	< 140 msec
S/D	< 0.50
A_{REV}	≥ 35 msec (peak velocity)
A_{REV}/A	≥ 0.6 (peak velocity or TVI duration)

LVEDP = left ventricular end diastolic pressure; LAP = left atrial pressure;
PCWP = pulmonary capillary wedge pressure; E = early transmitral filling fraction
A = late transmitral filling fraction (atrial contraction); DT = Deceleration time
S = Pulmonary venous flow during systole; D = Pulmonary venous flow in diastole

A_{REV} = Pulmonary venous reversal flow during atrial

Transmitral propagation velocity (pv; cm/sec) is a measure of the slope of the acceleration phase of the early diastolic transmitral flow (E wave) obtained by simultaneous application of color Doppler and M-Mode echocardiography. The propagation velocity is measured from the onset of early transmitral flow at the level of mitral valve annulus to approximately 4 cm into the ventricular cavity. Normal propagation velocity has been reported to be greater than 70 cm/sec, while values lower than 40 cm/sec are considered abnormal. This measure has been correlated with **diastolic function** as well as **chamber pressures**.

$$\textbf{PCWP} = 5.27 \times (E/pv) + 4.6$$

Another equation relating PCWP to pv is

$$\textbf{PCWP} = 4.5 \times \frac{10(3)}{[2 \times IVRT] + pv}$$

This equation has a specificity 100% and a sensitivity of 90% in predicting the PCWP of > 15 mmHg and has been shown to be load independent.

In patients with **atrial fibrillation** the use of regurgitant jets or flow patterns is problematic due to variability in flow velocities and the absence of transmitral and pulmonary venous 'A' waves. However, estimates of LAP and LVEDP can be made based on measurements of the transmitral **E wave peak acceleration** (PkAcc; cm/sec^2), **deceleration times** (DT; msec), and the **isovolumic relaxation time** (IVRT; msec).

$$\textbf{LVFP mmHg} = 22 + 0.005(PkAcc) - 0.183(IVRT)$$

$$\textbf{PCWP mmHg} = 51 - 0.26(DT)$$

Since the propagation velocity has also been shown to be a load independent measure, it may be a useful technique to estimate left sided pressures for patients with irregular rhythms.

Assessment of **pulmonary vascular resistance** (PVR) can be performed from the systolic time velocity intervals of the right ventricular outflow tract (RVOT) or main pulmonary artery (MPA) using either PW or CW Doppler from either esophageal or gastric windows. Normally, the pulmonary artery flow profile is symmetric. Asymmetry or notching suggest mild to moderately elevated, and severely elevated pulmonary vascular resistance respectively. Ratios of the time intervals of these flow profiles (RV pre-ejection period (RVPEP) seconds; Time Velocity Integral (TVI) cm) can be used to estimate pulmonary artery pressures

RVPEP/TVI	**< 0.4 seconds/M**	**= < 3 Wood units**
RVPEP/TVI	**= 0.4-0.6 seconds/M**	**= 3 – 7.5 Wood units**
RVPEP/TVI	**> 0.6 seconds/M**	**= > 7.5 Wood units**

The ratio of pre-ejection period (PEP) to the acceleration time (AT) is directly related to pulmonary artery systolic pressure.

PASP = PEP/AT

A ratio = 1 approximated a systolic pulmonary artery pressure of 25 mmHg or a mean pulmonary artery pressure of 15 mmHg. The relationship diverged with higher a higher ratio (> 1), however correlation improved when a logarithmic function was applied. This ratio divided by the total systolic time (TT; (PEP/AT)/TT) was also directly related to pulmonary vascular resistance.

PVR = -0.556 + 1.154 [(PEP/AT)/TT]

Additional Hemodynamic Assessments

Measures of pressure gradients and volumetric flows form the basis of quantitative assessments of a number of cardiac structures and pathologies including valvular disease and congenital defects. The **Bernoulli Equation** is commonly used to assess transvalvular gradients across stenotic lesions as in mitral and aortic stenosis. In addition, data has demonstrated that for mitral stenosis and aortic insufficiency the rate of decline in the stenotic or regurgitant pressure gradient (**pressure half time (PHT; P1/2)**) is related to severity of valve disease. Mitral valve area (MVA) can be calculated from the PHT based on empirically derived equation.

$MVAcm^2$ **= 220/PHT (msec)**

The PHT may be used to assess the severity of aortic insufficiency.

Mild AI **= PHT > 450 msec**
Moderate AI **= PHT 250-450 msec**
Severe AI **= PHT < 250 msec**

Intracardiac shunts can be assessed using similar calculations of stroke volume (SV) and cardiac outputs. The shunt volume may be calculated from the TVI of the shunt and the CSA of the defect. Since the defect may not be circular this measure may be erroneous. However, measuring left (LVOT or AoV) and right sided (MPA or RVOT) cardiac outputs the Qp:Qs can be calculated.

$$Q_p/Q_s = SV_{\text{Right Heart}}/SV_{\text{Left Heart}}$$
$$Q_p/Q_s = SV_{\text{MPA or RVOT}}/SV_{\text{LVOT or AoV}}$$

When combined with two-dimensional and color Doppler echocardiography, a complete assessment of congenital lesions is possible.

The principle of **conservation of mass** is the basis of the **continuity equation**. The continuity equation is an extension of the equation used to calculate volumetric flow. Assuming that mass or volume can be measured, the continuity equation states that, in the absence of intervening channels, the mass or volume at one location is equal to the mass or volume passing through another site. This assumes a circular tube through which blood flows with a constant diameter.

$$Mass_1 = Mass_2$$
$$Area_1 \times TVI_1 = Area_2 \times TVI_2 \qquad Area = \pi r^2 = \pi(D/2)^2$$

The **continuity equation** is commonly employed to measure the aortic valve (AoV) area (AVA).

$$Area_{AoV} = \pi(D_{LVOT}/2)^2 \times (TVI_{LVOT}/TVI_{AoV})$$

Another use of the **continuity equation** is seen during assessments using measurements of the **Proximal Isovelocity Surface Area** (PISA). Although PISA is more commonly referred to during assessment of mitral regurgitation, it has also been applied to stenotic lesions and congenital defects.

Conclusion

Doppler techniques provide a wealth of hemodynamic information that is critical to the diagnostic utility of echocardiography.

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NOTES

Advanced Echocardiography III

7:00 – 7:45 am Continental Breakfast with Exhibitors

7:45 – 8:00 am **Announcements**
GS Hartman/SK Shernan

Advanced Mitral Valve

Moderator: BA Bollen

8:00 – 8:30 am **Your Patient is Scheduled for Mitral Valve Repair but Presents with Only 2+ Regurgitation by Intraoperative TEE: What Now?**
SN Konstadt

8:30 – 9:00 am **Ischemic Mitral Regurgitation**
S Aronson

9:00 – 9:30 am **Surgical Repair of the Mitral Valve**
Surgeon (TBA)

9:30 – 10:30 am **Case Discussions with Faculty**
BA Bollen, SN Konstadt, S Aronson, Surgeon TBA

10:30 – 11:00 am Coffee Break with Exhibitors

Role of Echocardiography in Perioperative Clinical Decision-Making: Part I

Moderator: RM Savage

11:00 – 11:30 am **Role of Echocardiography in Percutaneous Approaches to Valve Dysfunction**
RM Savage

11:30 – 12:00 noon **Mitral Valve Dysfunction after Mitral Valve Repair**
SK Shernan

Luncheon Series (box lunch served)

12:00 – 12:15 pm Box Lunches Served

12:15 – 1:00 pm Surgeon (TBA)

Role of Echocardiography in Perioperative Clinical Decision-Making: Part II

Moderator: RM Savage

1:00 – 1:30 pm **How to Use Echocardiography to Help Wean from Bypass**
TM Burch

1:30 – 2:00 pm **Unanticipated Ascending Aortic Dilatation: How Big is Too Big?**
AT Cheung

2:00 – 2:30 pm **Echocardiographic Assessment of Myocardial Viability**
M Swaminathan

2:30 – 3:00 pm **Echocardiography for Adult Congenital Heart Disease**
IA Russell

3:00 – 3:30 pm **Stump the Panel: Unusual Cases from the Faculty**
RM Savage, TM Burch, AT Cheung, M Swaminathan, IA Russell

3:30 – 4:00 pm Coffee Break

"Test Yourself"

Moderator: DA Zvara

4:00 – 5:30 pm **"Test Yourself" Mock Test**
Faculty: DA Zvara, BC Spence, TM Burch, EG Avery, CG Koch

5:30 – 7:30 pm Dinner on Own

7:30 – 9:30 pm **Mock Exam Review**
Faculty: DA Zvara, BC Spence, TM Burch, EG Avery, CG Koch

NOTES

Your Patient is Scheduled for Mitral Valve Repair but Presents with Only 2+ Regurgitation by Intraoperative TEE: What Now?

Steven Konstadt, MD, FACC, MBA
Professor and Chairman
Department of Anesthesiology
Maimonides Medical Center
New York, NY

This presentation will discuss important considerations in the intraoperative assessment of mitral regurgitation. Emphasis will be placed on the determination of the severity of the MR and the mechanism of the MR and the proper intervention. The determination of the severity is primarily based on the anatomic and kinetic findings as seen by two dimensional imaging. The determination of severity is based a combination of color and spectral Doppler modalities and the physiologic state. The proper intervention is based on a complex assessment of the patient's condition.

MITRAL REGURGITATION

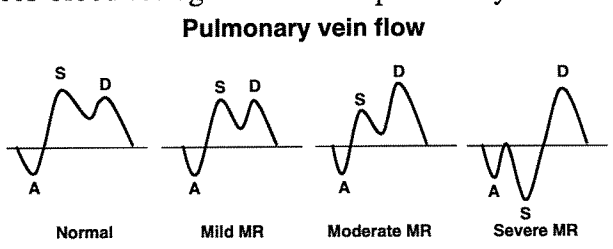
Pathology involving any or all of the structures comprising the mitral valve apparatus can cause mitral regurgitation. Myxomatous degeneration leading to leaflet prolapse is the most common etiology of mitral regurgitation requiring surgical correction. Chordae tendinae become elongated or rupture. One or both leaflets become redundant and prolapse or flail into the left atrium during systole. Infectious endocarditis causes leaflet perforation or scarring, or chordal rupture. A more ominous finding is an annular abscess indicated by an echolucent region or "pocket" adjacent to the annular attachment of the infected leaflet(s). This finding is a significant problem for the surgeon who needs strong viable tissue in which to sew a prosthetic valve. Paravalvular leaks may occur in the region of an annular abscess after valve replacement. Mitral regurgitation due to rheumatic endocarditis results from leaflet scarring and retraction, and chordal fusion. Annular dilatation is another cause of mitral regurgitation. As previously mentioned, the annulus dilates coincidently with ventricular dilatation, except when mitral annular calcification is prominent. The regurgitant jet tends to be centrally directed if annular dilatation is the sole reason for valvular incompetence.

Color flow Doppler is used to detect and quantitate regurgitant lesions by placing the color sector over the mitral valve and left atrium. Several methods are used to grade the severity of mitral regurgitation. Spatial area mapping of the color Doppler regurgitant jet provides an estimate of the degree of mitral regurgitation by measuring the regurgitant area or expressing the regurgitant area as a percentage of the left atrial size.[6] Severity is graded as trace, mild (1+), moderate (2+ to 3+), or severe (4+) (Table below).

Degree of MR	% jet area/LA area	Jet length
Trace	0 to 10%	< 1 cm
1+ (mild)	10 to 20%	1 to 2 cm
2+ (moderate)	20 to 30%	One third LA antero-posterior distance
3+ (moderate)	30 to 40%	1/3 to 2/3 LA antero-posterior distance
4+ (severe)	> 40%	> 2/3 LA antero-posterior distance

It is important to use multiple imaging planes and approaches to evaluate jet area, particularly if the jet is eccentric or is directed along the left atrial wall. Spatial area mapping of wall hugging jets underestimates the severity of regurgitation because energy is dissipated and velocity is reduced to a greater degree than centrally directed jets, given the same regurgitant orifice size and regurgitant volume. It is common practice to increase the estimated severity of wall hugging jets by one degree from the assessment made by spatial area mapping alone. This limitation emphasizes the need for several methods of assessment. Twenty-two signs of severe mitral regurgitation have been described.[7]

Pulmonary vein flow interrogation by pulsed wave Doppler echocardiography is another method of quantifying the degree of mitral regurgitation (Figure below).[8-10] The absence of valves in pulmonary veins allows transmission of left atrial and mitral regurgitation hemodynamics into the pulmonary venous system. The "A" wave, coincident with the "p" wave on the ECG, occurs because the atrial contraction that forces blood into the left ventricle through the mitral valve, also forces blood retrograde into the pulmonary veins. With the onset of left ventricular systole, nd blood travels antegrade in the pulr ces the "S" wave on the spectral Dopp lood moves from the left atrium to t ade through the pulmonary veins to spectral Doppler display. The S wa are in the same direction from baseline.



Mitral regurgitation (MR) changes the appearance of the pulsed wave Doppler analysis of pulmonary vein flow depending on the severity of MR. Mild MR causes blunting of the S wave, which becomes equal in height to the D wave. Moderate MR causes the S wave to be smaller than the D wave. Severe MR causes the S wave to occur in the opposite direction from the D wave (systolic reversal of pulmonary vein flow) as shown in the figure.

Mitral Valve Repair

In mitral valve repair, determination of the mechanism is often as important as the determination of the severity. The mechanism, not the etiology dictates the surgical actions needed to correct the problem. Dr Carpentier has devised a classification system based on leaflet motion. Here is the system:

Type I Normal Valve Function:-

- Annular Dilatation
- Leaflet Perforation

Type II – Excessive Leaflet Motion

- Prolapse
- Chordal rupture
- Chordal elongation
- Papillary muscle rupture

Type IIIA-Restricted Leaflet Motion

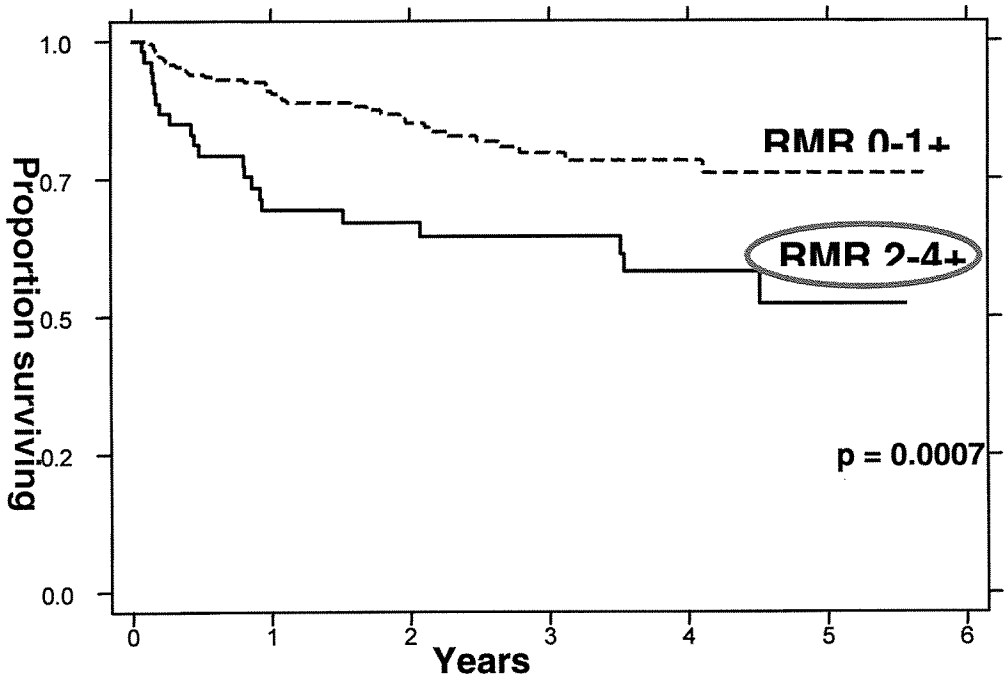
- Valvular/subvalvular thickening

Type IIIb – Restricted Leaflet Motion

- Papillary Muscle Displacement

Ischemia and MR

- 20 to 40% of coronary artery bypass grafting (CABG) candidates may also have chronic ischemic mitral insufficiency
- Significant residual mitral regurgitation after revascularization is associated with increased immediate postoperative and long-term morbidity and mortality



- Intraoperative hemodynamic changes complicate the assessment of the severity of residual MR.
- Intraoperative TEE evaluation is critical in order to determine the mechanism of MR and guide a strategy for repair
- Surgical repair of MR most commonly requires a downsized annuloplasty ring, in order to enhance coaptation, works satisfactorily in most cases of functional IMR

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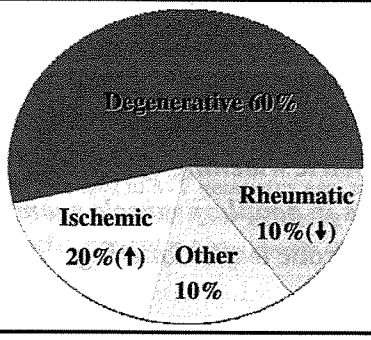
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ISCHEMIC MITRAL REGURGITATION

SOLOMON ARONSON, M.D.
FACC, FACP,FAHA,FASE

Professor Duke University
Executive Vice Chair Dept Anesthesiology

MITRAL VALVE DISEASE

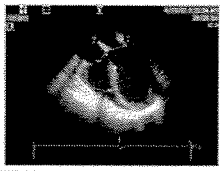


DILEMA OF ISCHEMIC MR

Inconsistent Definition

Structural Pathology often doesn't exist

Questionable Mechanism



Ischemic MR

Develops after MI *(15- 20%)*

Alters prognosis medical therapy

High operative mortality

Circulation 78:151-9,1988
Circulation 96:827-33,1997
Circulation 103:1759-64,2001
Circulation 111:254-6,2005

MV Replace or Repair Dilemma
Mortality/Morbidity

- **10-30% CABG asc. with IMR, (5% severe)**
- **15-50% mortality with MV- CABG**
- **15% (30 d) MV replacement**
- **10-30% Residual MR with annuloplasty alone**

J Heart Valve Disease 11:11-9,2002
J Thorac Cardiovasc Surg 128:916-24,2004

IMR assoc with worse prognosis

Grigioni et a. (Circulation 103:1759-11764, 2001)

- **Retrospective Review (patients S/P MI)**
- **Quantitative Doppler**
- **Long term outcome (5 years)**
- **Total Mortality and Cardiac Mortality**
- **Patients with IMR (62%, 50%)**
- **Patients No IMR (39%, 30%)**

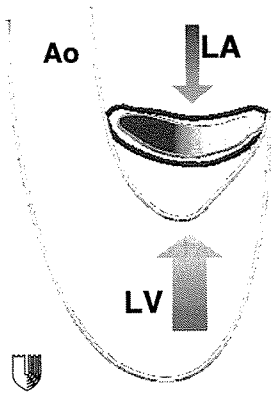
IMR ACUTE INFARCTION MECHANISM

-Unbalanced *ventricular forces*
-Expansion *borderzone myocardium*
-Asymmetric *widening of annulus*
-Changes in *papillary muscle geometry*
-Early systolic *leaflet loitering*

ISCHEMIC MITRAL REGURGITATION

(Dent et al, Am J Physiol 296;2100-2108,1995)

- Acute Ischemia (Dogs)
- 2D-Echo/LAP-LVP measurements
- Diastolic LAP –LVP Gradient determines MV leaflet opening
- Ventricular Elastic Recoil determines MV leaflet closure



Saddle shape valve

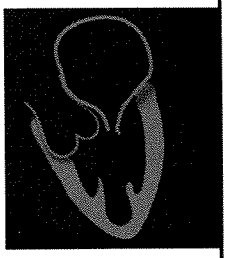
More force required To move leaflets cephalad than caudally

If LVP-LAP gradient is low, then MR

GEOMETRY - DYNAMIC RELATIONSHIP

PAP TIP – ANNULUS
constant (normal hearts)
not constant (ischemic hearts)

PAP TIP - LEAFLET EDGE
constant (normal & ischemic)

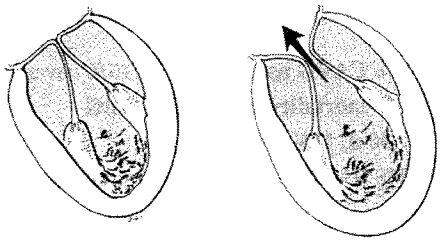


ISCHEMIC MITRAL REGURGITATION

Komeda et al (Circulation 96; II 128-133, 1997)

- Acute ischemia (dogs)
- Radiopaque markers/CFD
- Radial dislocation PMPM asc. with MR
- Apical motion (tethering) of PML
- Apical & Radial displacement additive
- Annuloplasty augments coaptation





JACC 1992

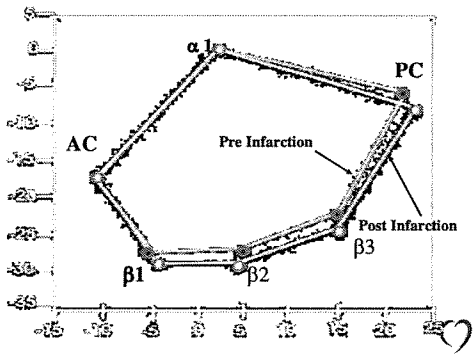
ANNULUS

- Annulus area decreases 25% during systole
- Most changes in annulus are posterior
- Posterior MI causes poor contraction of posterior annulus (*theory*)

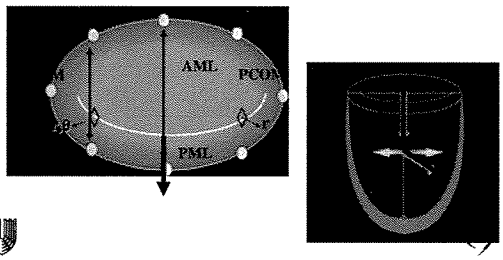
ISCHEMIC MITRAL REGURGITATION

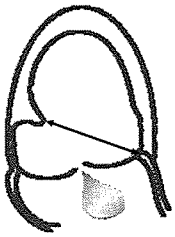
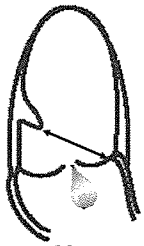
Gorman et al (Ann Thoracic Surg 64:1026-31, 1997)

- Acute infarction (sheep)
- Sonomicrometry
- Small changes in MV geometry asc. with severe MR, if subvalvular tethering & leaflet restriction present *(following posterior MI)*



Posterior annular dilation predominance



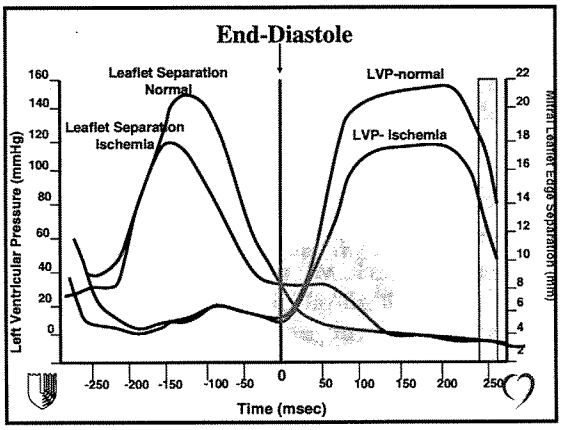
Inferior MI	Anterior septal MI
	
Annular Size:	Comparable
LV Dilation:	Anteroseptal > Inferior
*PMPM Tethering Distance:	Inferior > Anteroseptal

*Strongest predictor

ISCHEMIC MITRAL REGURGITATION

Glasson et al (J Thorac Cardiovasc Surg 116;193-205, 1998)

- Acute Ischemia (sheep)
- Radiopaque Markers / Biplane Videofluoroscopy
- Dynamics throughout cardiac cycle
- Incomplete coaptation end diastole/early systole
- Delayed closure both leaflets
- Asc. with larger annular area (end-diastole)
- "Loitering" when LV geometry unchanged



ISCHEMIC MITRAL REGURGITATION

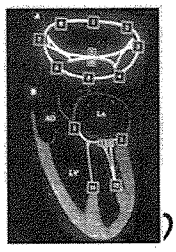
Timek et al (J Thorac Cardiovasc Surg 119; 774-83, 2000)

- Acute ischemia (sheep)
- Radiopaque markers / Biplane videofluoroscopy
- Ring annuloplasty subgroups
- MR asc. with Coaptation delay
- MR asc. with increase ED annular diameter
- No MR ,annuloplasty group (*eliminated delayed coaptation*)

ISCHEMIC MITRAL REGURGITATION

Dagum et al (Circulation; 102:II 62-69, 2000)

- Acute ischemia (sheep)
- Radiopaque markers/CFD
- Effects of annuloplasty tested
- Increase pap-annular distance asc. with IMR
- Ring annuloplasty preserved pap-annular distance



RING ANNULOPLASTY

- Decrease annulus area
- Alters position & dynamic of papillary tip
- Alters leaflet coaptation dynamic

(changing papillary muscle position)

IMR

- Standard surgical repair is to undersize the annuls in order to increase leaflet coaptation

IS THAT ENOUGH

J Thorac Cardiovasc Surg 122:1059-62,2001
J Thorac Cardiovasc Surg 122:1125-41,2001

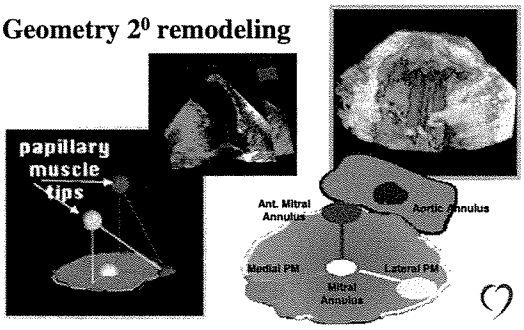
ISCHEMIC MITRAL REGURGITATION

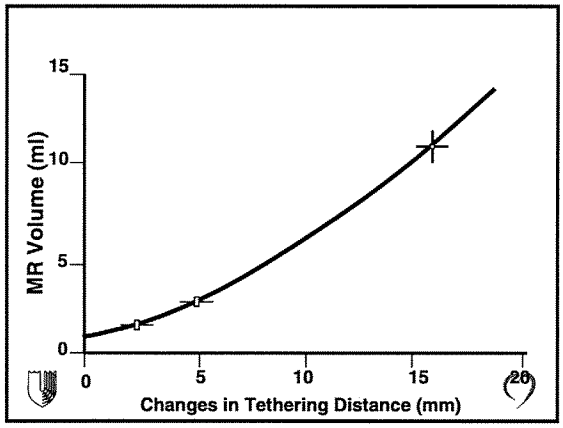
Leil - Cohen, et al (Circulation 101:2756-63, 2000)

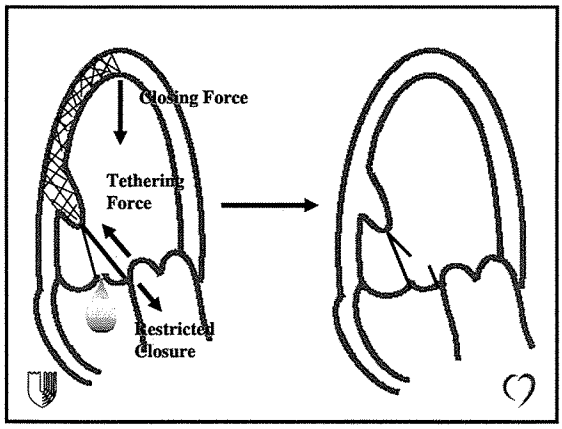
- Chronic infarction (sheep)
- 3D Echo
- PM tip to annulus distance examined
- Curvilinear relationship between tethering distance and MR severity
- IMR related to LV geometric changes

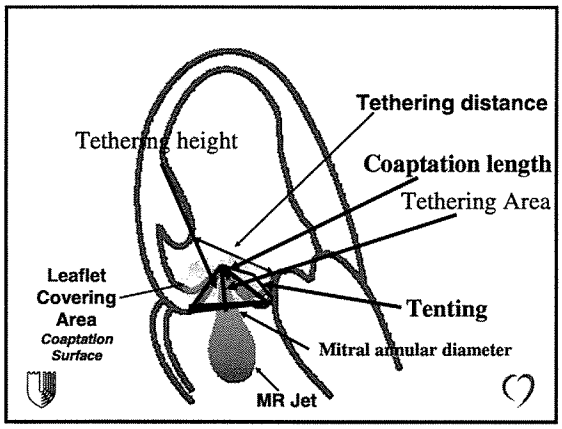
3D ECHO ASSESSMENT

Geometry 2^0 remodeling









If coaptation depth > 11mm, then Mitral Annulus Size may not be limiting

Thus Ring Annuloplasty inadequate & LV plication or realignment of Papillary muscle geometry needed to preserve valve.

Ann Thorac Surg. 2001; 71:1146-53

ISCHEMIC MR

(MECHANISM)

Deformation of Annulus

PM dynamic disturbance

LV shape and contractility change

LV and MV integration

Abn. geometric relationship

ISCHEMIC MR
(TREATMENT)

- **Coronary revascularization**
- **Ring annuloplasty**
- **Valve repair / replacement**
- **LV reconstruction**
  - **Localized Reshaping (*LV plication, off CPB*)**
  - **Resecting post wall muscle (*between PM, CPB*)**

IMR *Repair or Replace*

Depends on:
MR pathophysiology
Patients clinical status
Lateral wall motion abn.
Apical leaflet tenting
Acute MI
Emergency
Rupture pap.

Eur J Cardiothorac Surg. 1995 Cohn
J Cardiothorac & Vasc Surg .2001 Grossi
J Cardiothorac & Vasc Surg .2001 Gillinov

IMR *Repair or Replace*

Annuloplasty > 50% failure if:
Annular diameter > 3.7cm (4 ch view)
Coaptation depth > 11mm
Tenting area > 1.6cm^2 (long axis view)
MR grade > 3+

Eur J Cardiothorac Surg. 1995 Cohn
Ann Thorac Surg. 2001; 71:1146-53, Calafiore
Am J Cardiol 98;50-8,2006
J Cardiothorac & Vasc Surg .2001 Grossi
J Cardiothorac & Vasc Surg .2001 Gillinov

MVR or Repair
Benefit mitigated if

LIMA not done
Lateral RWMA present
MR jet complex

IMR DECISION ANALYSIS

Patient Symptoms

Angina → CABG
CHF → Valve + CABG

+ ECHO / Symptoms

Mild MR → CABG
Mod MR
- Good LV&RV, No Afib, Sm LA → CABG
- Bad LV or RV, Afib, Lg LA, PFO, other pathology → CABG + Valve

Severe MR → CABG + Valve

IMR SUMMARY

Heterogeneous population
50% 5 yr survival
Repair vs MVR decision multifactorial
Pathogenesis of leak is important
- **Reversible inferior RWM (viable)**
- **Lateral RWM**

Role of Echocardiography in Percutaneous Approaches to Valve Dysfunction

RM Savage

Percutaneous MV Repair & AVR
Echocardiographic Evaluation

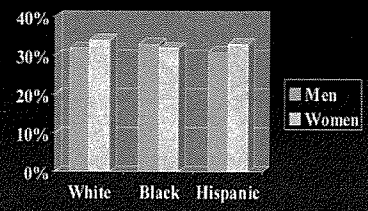
Robert M. Savage, M.D., F.A.C.C.
Head, Section of Perioperative Echocardiography
Cardiothoracic Anesthesia and Cardiovascular Medicine
The Cleveland Clinic Foundation
Cleveland, Ohio USA

Goals of Management
TCD Procedures

- Insure patient safety
- Protect technology
- Protect institution

Percutanerous MV Repair

Prevalence of Mitral Regurgitation in the Elderly



Aronow WS et al. Am J Cardiol 2001;87:1131-3

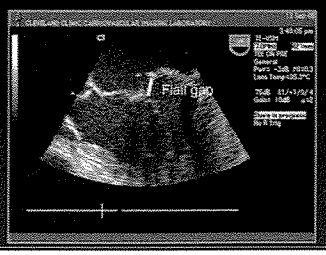
Critical Issues in Transcutaneous MV Repair

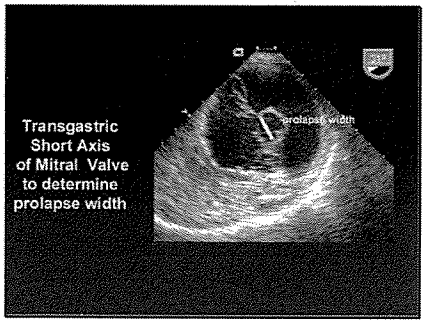
- Confirm Diagnosis and Repairability
  - Pathology and segmental abnormality
  - Severity of Pathophysiology
- Procedural Guidance
  - Intra-atrial septal puncture
  - Positioning over abnormal segment
  - Capture Path Alignment
  - Change in MR Severity
  - Need for Second Clip
  - Device withdrawal
- Complications
  - Septal puncture
  - Trauma with deployment catheter
  - Unsuccessful repair
  - Valve apparatus trauma
  - Hemodynamic Instability
  - Malve stenosis

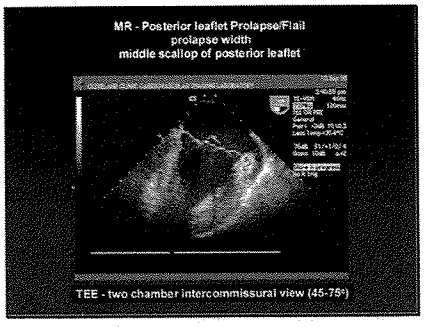
Perioperative TEE
Guidance of Percutaneous Mitral Repair

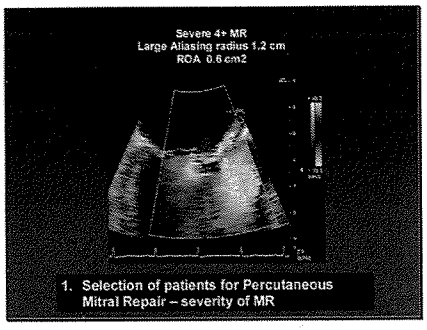
1. Patient selection
2. Transseptal crossing
3. Alignment of delivery system perpendicular to mitral plane in center of coaptation line
4. Alignment of open device with arms perpendicular to coaptation line
5. Closing of arms and grasping mitral valve
6. Release of implant device
7. Assessing severity of MR compared to before
8. Follow up

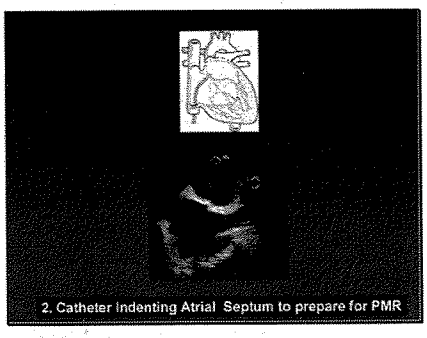
MR - Posterior leaflet Prolapse/Flail
Long axis 0° plane

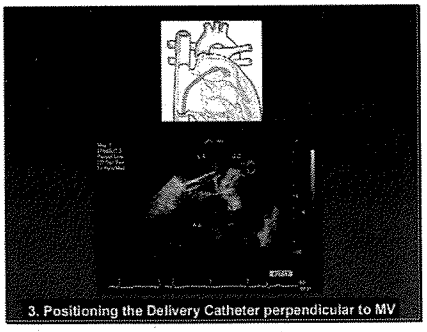


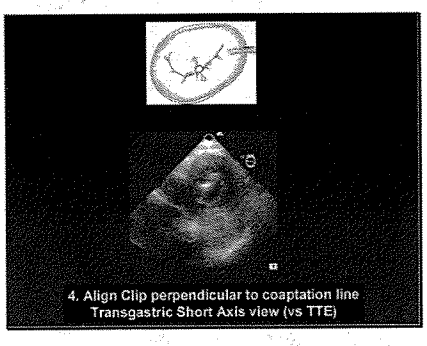


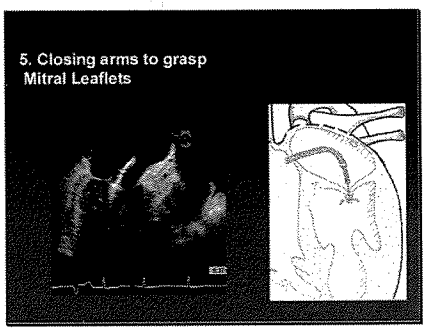

MR - Posterior leaflet Prolapse/Flail
prolapse width
middle scallop of posterior leaflet
TEE - two chamber intercommissural view (45-75°)

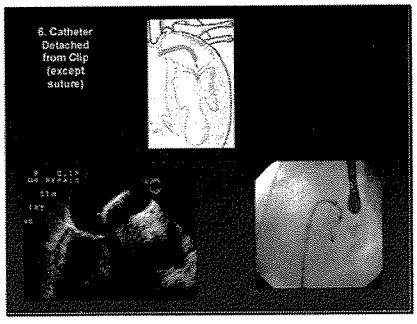

Severe 4+ MR
Large Aliasing radius 1.2 cm
ROA 0.6 cm2
1. Selection of patients for Percutaneous Mitral Repair – severity of MR

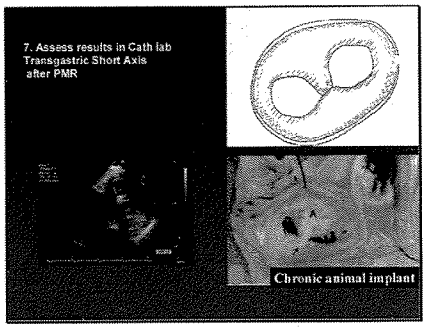

2. Catheter Indenting Atrial Septum to prepare for PMR


3. Positioning the Delivery Catheter perpendicular to MV


4. Align Clip perpendicular to coaptation line
Transgastric Short Axis view (vs TTE)


5. Closing arms to grasp
Mitral Leaflets


6. Catheter
Detached
from Clip
(except
suture)


7. Assess results in Cath lab
Transgastric Short Axis
after PMR
Chronic animal implant

Severe 4+ MR
Before PMR

8. Transthoracic
Echo follow up
after Percutaneous
Mitral Repair (PMR)

Complication Management PMVRep

Unsuccessful Repair
MV Apparatus Trauma
Clip Detachment

Percutaneous Aortic Valve

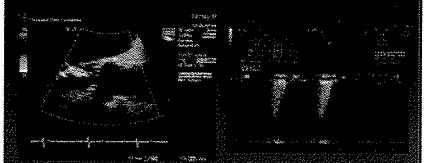
Critical Issues in Transcutaneous AVR

- Confirm Diagnosis and Severity
- Procedural Guidance
  - Pacing wire placement
  - Transaortic wire placement
  - Baloon Aortic Valvuloplasty
  - Aortic Root Alignment of Device
  - Coronary Ostia Patency
- Complications
  - Aortic trauma
  - Coronary embolization
  - Coronary Occlusion
  - Mitral valve dysfunction (antegrade)
  - Post-deployment regurgitation
  - Post-Deployment Gradient
  - Device Embolization

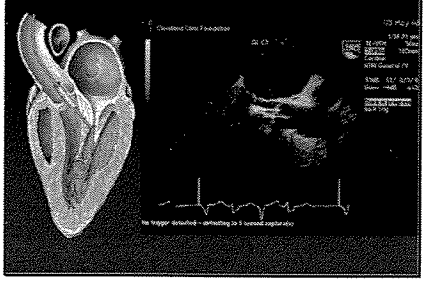
1. Confirm Diagnosis and Severity



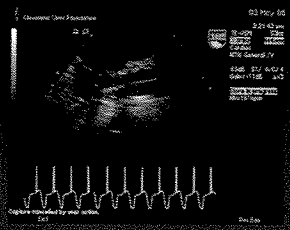
1. Confirm Diagnosis and Severity



2. Device Guidance Baloon Valvuloplasty



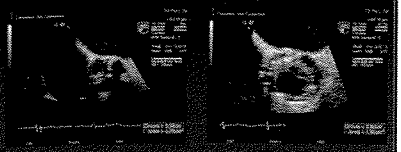
3. Device Deployment- Positioning



4. Confirmation of Results

Before

After



Percutaneous Aortic Valve

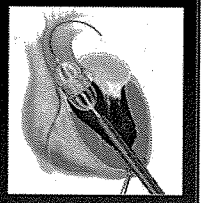
Before

After



Transapical Aortic Valve Replacement

- New technology for minimally invasive aortic valve replacement



Complication Managemenet Retrograde PAVR

Prosthetic Leaflet Restriction
Trauma MV
Left Main Occlusion
Valve Embolizatoin

The Greatest Risk .... Not Taking One.

Perioperative Echocardiography for Mitral Valve Repair

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Director of Cardiac Anesthesia
Department of Anesthesiology, Perioperative and Pain Medicine
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Harvard Medical School

Over the last several years, the impact of TEE's influence on perioperative cardiac surgical decision-making has become increasingly more appreciated. TEE, however, has been suggested to be particularly useful in the perioperative evaluation of mitral valve (MV) disease. Early studies commented on the use of epicardial echocardiographic scanning of the MV on the field by the surgeon (**1**). However, because of the MV's orientation in relation to the esophagus, excellent intraoperative TEE windows for two-dimensional (2-D) and both qualitative and quantitative Doppler evaluation can easily be obtained. Accordingly, TEE has supplanted surgeon-guided epicardial scanning for evaluation of the MV (**2**).

A thorough TEE examination of the MV requires the utilization of all echocardiographic modalities. In the initial pre-cardiopulmonary bypass (CPB) period, 2-D echocardiography defines the extent and etiology of MV pathology. The severity and location of MV disease including subvalvular involvement, annular calcification and dilation, bi-leaflet prolapse and anterior mitral leaflet (AML) disease, along with the degree of left ventricular (LV) function, should all be assessed as this information when integrated together, can be helpful in determining whether MV replacement (MVR), repair, or neither is indicated. It is also important to diagnose if existing severe mitral regurgitation (MR) is primarily associated with ischemia, as opposed to rheumatic or myxomatous disease, since the extent of surgical treatment for ischemic MR remains controversial. Although the 2D anatomic images and the color flow Doppler (CFD) modalities are used most often during interrogation of the MV in the operating suite, a complete echocardiographic evaluation using pulse wave (PWD) and continuous wave Doppler (CWD) and even three-dimensional echocardiography (3-D) can assist in determining the severity of blood flow disturbances associated not only with MR, but with mitral stenosis (MS) (**3**).

Clinical studies have suggested that the *pre-CPB* TEE exam prompts changes in surgery in 9-14% (**1,2**) of patients undergoing MV surgery. The population of patients influenced by the pre-CPB TEE exam includes those initially scheduled only for coronary bypass grafting (CABG) who also require a MV procedure. In addition, the pre-CPB exam also influences surgical decision-making in those patients scheduled for an MV procedure based upon the preoperative transthoracic echocardiographic (TTE) or TEE exam, who actually only require a CABG due to "improvement" in the severity of MV disease as determined by the pre-CPB TEE examination in the operating room. The influence of changes in hemodynamic loading conditions during general anesthesia on trans-mitral flow and pressure, the potential changes in the patient's overall medical

condition since the time of the pre-operative echocardiographic exam, the technical limitations of TTE in comparison to TEE and the general subjectivity of the assessment among different echocardiographers, must all be taken into consideration when making the final surgical decision regarding the most appropriate MV procedure.

The initial post-CPB TEE exam is essential in helping to determine the competency of the replaced or repaired MV, persistent MR and/or systolic anterior motion (SAM). It is also important for the echocardiographer to fully understand normal flow patterns and acceptable transvalvular pressure gradients. Most small perivalvular jets detected by CFD are insignificant, although larger "leaks" may become associated with hemolysis, hemodynamic instability or valvular dehiscence. Hemodynamic loading conditions and LV function must also be taken into consideration in the assessment of residual MR. Following MV procedures, several studies have suggested that on the average, in approximately 5 -11% of cases **(1,2, 4-6)** the post-CPB TEE exam may identify persistent lesions that require additional, immediate surgical intervention.

Persistent MR following MV Repair:

It is clear from the studies cited that intraoperative echocardiography can provide the surgeon with an immediate evaluation of MV function after repair. Moderate to severe residual MR will usually necessitate a return to CPB for further evaluation and definitive surgery. One main question arises regarding MV repairs and the utility of intraoperative echo: What is the outcome of patients who have trace to mild residual MR after repair?

One study attempting to address this topic included 76 patients with residual "1+ or 2+ MR" following MV repair in whom the surgical team decided not to return to CPB **(7)**. These patients were compared to a control group matched for preoperative risk who also underwent MV repair without evidence of residual MR. Follow-up data was available for an average of 3 years for each group. There was no difference in morbidity (congestive heart failure, embolic events, surgical mortality) between the 2 groups, however, re-operation for a second MV procedure was required in only 3 of the "no MR" group compared to 9 patients in whom persistent MR was initially demonstrated. The authors concluded that insignificant residual MR should not raise excessive concern but expressed caution about the trend toward re-operation.

Systolic Anterior Motion Following Mitral Valve Repair:

Following MV repair, the presence of left ventricular outflow tract (LVOT) obstruction has typically been associated with systolic anterior motion (SAM). Risk factors for SAM include *preoperative* evidence of excessive or redundant posterior (PML) and/or AML tissue, non-dilated LV and a narrow mitro-aortic angle or *post-operative* evidence of persistent AML redundancy, excessive PML resection, the selection of an annuloplasty ring that is either too small or incorrectly oriented, and an hypertrophied/hyperdynamic LV all of which contribute to a Venturi effect that "pulls" the AML and coaptation point toward the ventricluar septum during systole.

The mechanism of SAM post MV repair may not be as simple as a "Venturi" effect on the AML, but instead may involve a more complex relationship between the MV leaflets and adjacent ventricular structures. Maslow et al studied the intraoperative TEE in 11 patients who developed SAM post MV repair in an attempt to determine if any echocardiographic findings were common **(8)**. Off line measurements included mitral annular diameter, PML and AML lengths, and distance from the coaptation point of the

MV to the ventricular septum. These measurements were compared to 22 matched control patients who underwent MV repair without evidence of SAM. Pre-repair, patients who displayed SAM post MV repair had a shorter anterior leaflet (2.13 ± .34 cm to 2.68 ± .39 cm), a longer posterior leaflet (2.18 ± .37 cm to 1.42 ± .27 cm), a shorter AML/PML ratio (0.99 ± .14 vs 1.95 ± .27) and a shorter distance from coaptation point to the ventricular septum (2.53 ± .34cm vs 3.01 ± .56cm). Surprisingly, the mitral annular diameters were not different in each group. In an illustration of the complexity of the mechanism of SAM post repair, the lower AML/PML ratio implied that a relatively greater contribution of the PML to the coaptation of the MV, which may predispose to SAM post repair. Additionally, a shorter distance between the valvular coaptation point and the ventricular septum may also predict SAM following MV repair.

Another echocardiographic study comparing 8 patients who developed SAM **(9)** with a control population who did not develop SAM post repair found that the AML was significantly longer than the AML (3.5 ± .40cm vs 2.8 ± .32 cm) and the ratio of the AML/PML to be almost identical. In this investigation, however, measurements of the mitral leaflet lengths were performed in mid-diastole whereas in the previous study, the mitral leaflet lengths were measured at the start of ventricular systole making a comparison between the studies somewhat difficult.

Several studies have identified SAM as a contribution to the requirement for early and late MV reoperation **(10,11)**. Freeman et al **(11)**, reported on 143 MV procedures for MR associated with myxomatous degeneration. Over 9% of these patients demonstrated echocardiographic evidence of SAM following MV repair. Although all of these patients had PML prolapse, 7 also had AML prolapse. The majority underwent PML resection (N=10) and 12 underwent ring annuloplasty (Duran =10, Carpentier=2). In this investigation, reinstitution of CPB was required in 5 patients due to persistent 3- 4+ MR which subsequently resolved following the second procedure. In the remaining 8 patients, SAM and concurrent MR was associated with hypovolemia and a relative hyperdynamic heart. Resolution of hemodynamic instability and pathology was achieved with volume resuscitation and reducing the administration of inotropic therapy. Marwick et al **(10)** reported that 10/309 mitral patients were observed to have LVOT obstruction and all 10 required reinstitution of CPB for either removal of the MV ring (N=5) or MVR (N=5).

In the absence of an echocardiographic evaluation, the diagnosis of post-MV repair SAM and its treatment, can be challenging since commonly employed post-CPB therapeutic interventions such as the use of inotropic agents, may actually be relatively contraindicated and lead to further hemodynamic compromise. Thus, it is essential to include a comprehensive evaluation for the presence of SAM in the post-CPB TEE examination following MV repair. The echocardiographic evaluation of SAM following MV repair includes a 2-D assessment of the mitral apparatus, a qualitative and quantitative CFD analysis of the MR jet and a quantification of the pressure gradient across the LVOT obstruction. The CFD jet is typically directed toward the left pulmonary vein and away from the atrial septum as the AML is pulled toward the LVOT. Obstruction in the LVOT can produce pressure gradients as high as 100 mm Hg. Typically the Doppler flow velocity profile associated with dynamic LVOT obstruction can be differentiated from the profile associated with aortic stenosis (AS) by its "dagger-shape" and mid-systolic acceleration. It would seem reasonable to assume that in the presence of echocardiographic confirmation of SAM and a hypovolemic and/or

hyperdynamic LV, a trial of volume resuscitation and reduction of intropic therapy as suggested by Freeman et al **(11)** is initially warranted, followed by a reevaluation of any persistent MR and LVOT obstruction before returning to CPB. Maslow et al **(8)** reported that SAM resolved completely in 4 patients with this treatment and, in another 4 patients, SAM was still present but without CFD evidence of LVOT obstruction or MR. The authors do not comment on whether the 3 remaining patients required further surgery.

Post-MV Repair Mitral Stenosis:

The initial post-CPB TEE exam is essential in helping to determine the competency of the repaired MV, persistent regurgitation and/or SAM and more rarely, acute MS. It is especially important for the echocardiographer to fully understand the normal flow patterns and acceptable transvalvular pressure gradients following various types of MV repairs. Chronic MS following repair may develop from thrombus, calcification or pannus formation, which restricts native leaflet function. Rarely, acute MS may develop due to technical limitations involving excessively restrictive MV annuloplasty rings, or stenotic edge-to-edge repairs.

Specific echocardiographic criteria for the diagnosis of early MS after MV repair have not been well established. Two-dimensional echocardiographic diagnosis of MS following post-MV repair may be difficult especially in those patients undergoing an Alfieri, edge-to-edge repair in which the mid-portion of the anterior and posterior leaflet are intentionally sutured together to prevent MR. In addition, classical Doppler echocardiographic measures for quantifying native MS such as the pressure half-time, may not be applicable immediately following MV valvotomy or repair due to acute changes in orifice geometry and chamber compliance. In addition, although some have suggested that significant residual MS following MV repair is defined by a persistent mean transvalvular gradient > 5 mm Hg or an area < 1.5 cm^2, the influence of concurrent MR and cardiac output must also be considered. Thus, the definitive diagnosis of residual MS following MV replacement or repair ultimately requires an integration of an in depth knowledge of the procedure, hemodynamics and a comprehensive intraoperative echocardiographic examination using all available modalities.

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NOTES

Utility of Echocardiography to Facilitate Weaning from Cardiopulmonary Bypass

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Educational Objectives:

1. Understand the anesthetic-hemodynamic considerations in successful weaning from CPB
2. Discuss the etiologies of myocardial dysfunction during separation from CPB
3. Discuss the use of intraoperative echo in guiding the separation from CPB
4. Discuss the therapeutic options available in difficult weaning from CPB and their indications

Transesophageal echocardiography (TEE) is a powerful, non-intrusive diagnostic tool that allows the anesthesiologist to serially evaluate the anatomy and function of the heart, pericardial space and thoracic aorta during cardiac surgery. The complex physiologic and hemodynamic alterations that occur during weaning form cardiopulmonary bypass (CPB) represent a unique challenge to anesthetic management. Successful separation from CPB requires attention to a number of cardiac and non-cardiac issues. These include adequate ventilation and oxygenation and restoring normal pH, potassium levels, and temperature. Cardiac factors include establishing adequate right and left ventricular contractility, preload, acceptable rhythm, absence of intracardiac air, and acceptable ventricular valvular function. TEE is helpful in assessing many of the cardiac factors needed to successfully wean from CPB and these will be discussed below.

CABG Surgery

Separation from CPB after coronary artery bypass grafting surgery (CABG) can be complicated by a variety of problems including: inadequate or excessive preload, ventricular dysfunction, air embolization, and new valvular abnormalities. Ventricular dysfunction can occur as a result of inadequate myocardial protection, myocardial ischemia due to inadequate revascularization, air embolism, and reperfusion effects such as myocardial edema and stunning. Untreated left ventricular dysfunction during weaning form CPB may result in new or worsening mitral regurgitation as the ventricle progressively dilates and fails. Similarly, right ventricular dysfunction during weaning from CPB may result in new or worsening tricuspid regurgitation, RV dilation and underfilling of the left ventricle. TEE provides dynamic information about right and left ventricular function, preload, adequacy of deairing, and valvular function which is helpful in achieving successful separation from CPB after CABG surgery.

Preload Assessment

TEE provides a qualitative estimate of right and left ventricular preload and volume status by observing changes in the ventricular end-diastolic area (EDA) as the heart is progressively filled and CPB support withdrawn. Because blood and fluid are non-compressible under physiologic conditions, ventricular end-diastolic chamber size correlates directly with volume status. The mid-esophageal four chamber view and the transgastric midpapillary short axis view are well suited for this purpose. TEE estimates of the end-systolic and end-diastolic area as a measure of ventricular volume correlate well with radionuclide estimates of ventricular volume. Clementes et al. (1) compared short axis images of the LV obtained by TEE to assess LV size to those obtained by radionuclide angiography (RNA). These authors found good correlation between TEE derived ESA and EDA as measures of LV volumes compared to RNA estimates. The conventional method for estimating LV preload is measurement of central venous or preferably pulmonary capillary wedge pressure. It is well known that these pressure measurements do not bear a linear relationship to measurement of LV end-diastolic volume because chamber compliance is typically not known, and often changes dramatically

during the course of cardiac surgery. Cheung et al. and others (2, 3) have shown that preload assessment by TEE using EDA is more accurate and clinically useful than estimates of preload based on hemodynamic data from a pulmonary artery catheter. When hypovolemia is suspected based on a reduction in EDA, response to therapy is readily assessed by noting the change in EDA as volume is administered. Conversely, over-zealous volume administration will be manifest as dilation of the LV with significant increases in EDA.

Systemic Vascular Resistance (SVR)

Hypotension due to low systemic vascular resistance will be indicated by a normal end diastolic area (EDA) and a low end-systolic area (ESA). This can be treated with appropriate alpha and/or vasopressin-1 (V-1) receptor agonists.

Contractility and Ejection Fraction

Qualitative estimation of LV and RV contractility and ejection fraction by TEE during weaning from CPB is accomplished by integrating the degree of endocardial wall motion, wall thickening, and the relative change between ESA and EDA as volume loading of the heart is undertaken. The mid-esophageal four chamber view is well suited for this purpose. Clementes et al. (1) demonstrated that echo-estimated ejection fraction using ESA and EDA correlated well with radionuclide measurement of ejection fraction. Progressive increases in ESA coupled with chamber dilation and poor wall thickening indicated diminished ventricular contractility. Increasing left ventricular failure may lead to new or worsening mitral regurgitation. One of the greatest benefits of TEE during weaning from CPB is the ability to detect ventricular dysfunction prior to complete separation from CPB to avoid a post-pump state of hypotension, low cardiac output, and ventricular dilation. Poor ventricular function detected during the weaning process can be due to the multiple factors listed above. Once poor contractility is noted by TEE, CPB support should be continued to allow time for diagnosis and treatment, i.e. further de-airing of the heart, reevaluation of bypass grafts etc. If contractility remains depressed, inotropic therapy and or intraaortic balloon pump support should be considered. The response to therapy can be gauged by looking for improved wall thickening and a decrease in ESA while the ventricle is gently volume loaded on CPB. If a balloon pump is needed, TEE can also help ensure it is properly positioned. The right ventricle is eccentric in shape and "wrapped around" the left ventricle making assessment of RV function challenging. Despite this, TEE can be used in a similar fashion to detect RV failure during weaning from CPB. The mid-esophageal four chamber view, and the transgastric mid papillary short axis view, are both helpful for this; however complete visualization of the RV requires multiple additional views. RV dysfunction due to ischemia, pressure, or volume overload results in RV chamber dilation and changes the normal pattern of septal curvature. In this setting, the LV EDA is often reduced with tricuspid regurgitation and intra-atrial septal bowing seen. Response to therapy aimed at treating RV failure can be assessed using TEE by watching for a decrease in RV chamber volume, improved RV free wall motion and a reduction in tricuspid regurgitation.

Segmental Wall Motion

Identification of new segmental wall motion abnormalities (SWMA) is readily accomplished during weaning from CPB by inspection of regional segmental wall thickening. The significance of mild changes in wall thickening such as a previously normal segment becoming hypokinetic is difficult to interpret. Multiple factors, such as myocardial edema, reperfusion effects, stunning, conduction abnormalities and translational wall motion, all reduce the specificity of mild SWMA changes. However, segments which previously had normal or mildly reduced function that become akinetic or dyskinetic after separation from CPB may indicate a significant problem. Leung et al. (4) studied the significance of regional wall motion abnormalities in patients undergoing CABG and found that persistent new SWMAs that occurred after CPB were associated with an increase in postoperative

myocardial infarction, ventricular failure, and death. In a subsequent study (5) a graded dobutamine infusion was used in the post CPB period to determine which new SWMA were due to ischemia, stunning or nonviable myocardium. This revealed the majority of post CPB SWMA were due to stunning and dobutamine stress echocardiography in the immediate post CPB period had low sensitivity in predicting nonviable myocardium. Nonetheless, the presence of new SWMA detected after CPB should be discussed with the surgeon and response to therapy, such as revision of a bypass graft to the affected region, monitored by serial reassessment of segmental wall thickening.

Intracardiac Air

TEE is exquisitely sensitive to the presence of intracardiac air and is an effective tool to ensure adequate deairing during separation from CPB. During open cardiac procedures and CABG surgery utilizing a single cross clamp technique, it is common to see intracardiac air bubbles. Larger air collections are potent specular reflectors typically seen in the LV apex or left atrium as bright "star bursts" image, while smaller collections of air appear as microbubbles "fire flies", often seen in the atrium, left atrial appendage, and pulmonary veins. Air embolism on the left-sided circulation can cause neurologic injury, transient ventricular dysfunction, and arrhythmias. A classic finding related to air embolization is acute right ventricular failure due to air embolization to the anteriorly located right coronary artery or saphenous vein graft. Echo-guided deairing can be accomplished by allowing the LV to eject while being vented with partial CPB support, and/or by direct aspiration of the air by the surgeon. A gradual clearing of microbubbles and the absence of larger air pockets indicate successful deairing. The process may require multiple "deairing trials" employing sequential changes in bed position with manual ventilation of the lungs and digital compression of the affected heart chambers by the surgeon. Multiple echo imaging planes are useful for this purpose.

Valvular surgery

TEE can be used in a variety of ways during separation from CPB following valvular surgery. These applications encompass key diagnostic functions of perioperative TEE that have been shown to benefit patient outcome (6-10) and compliment the previously discussed applications. When a mechanical or tissue bioprosthetic valve is implanted, TEE is essential to ensure normal leaflet or occluder motion and the absence of a significant paravalvular leak. Detection of impaired valvular function and paravalvular leakage during weaning from CPB may require multiple imaging planes and use of color and spectral Doppler modes. If a significant problem with a valve replacement is detected, adequacy of surgical correction can be assessed by repeating the echo evaluation when repair of the problem is completed. Once off CPB, baseline hemodynamic function of the bioprosthesis such as the mean and peak transvalvular gradient can be documented (11). Following mitral valve repair, TEE is used to detect and treat left ventricular outflow tract obstruction due to systolic anterior motion of the anterior mitral valve leaflet as CPB support is withdrawn. Once off bypass, the amount of residual mitral valve regurgitation can be quantified, mean transvalvular gradient measured and overall adequacy of mitral valve repair assessed.

Diastolic Function:

TEE allows rapid accurate assessment of global LV diastolic function by a combination of Doppler measurement of transmitral inflow velocities, pulmonary venous velocities, transmitral color M-mode propagation velocities and tissue Doppler measurement of the mitral annular velocities (13). Although it will not be the focus of this lecture, impaired diastolic function has been associated with increased mortality (13) and patients with impaired diastolic function will require higher filling pressures to achieve an adequate end diastolic volume.

In summary, TEE enables the anesthesiologist to directly observe heart function during the complex process of weaning patients from CPB. Direct visualization of ventricular chamber size and systolic wall thickening coupled with measurement of blood pressure enable early detection of ventricular dysfunction, estimation of preload and identification of new or worsening valvular function. Once a problem is detected during the weaning process, TEE can readily assess the success or failure of corrective therapy and indicate when it is appropriate to continue separation from CPB. When open cardiac procedures, such as valve repair or replacement are performed, TEE permits early detection of prosthesis dysfunction, inadequate repair or inadequate air removal.

Practice Questions:

Use the following table to answer the next four questions.

HD Scenario:	Wall Thickening	EDA	ESA	FAC	CO	E/E'
Scenario A	Normal	Decreased	Decreased	Normal	Decreased	< 15
Scenario B	Normal	Decreased	Decreased	Normal	Decreased	> 15
Scenario C	Normal	Normal	Decreased	Increased	Increased	< 15
Scenario D	Decreased	Increased	Increased	Decreased	Decreased	> 15
Scenario E	Normal	Increased	Increased	Increased	Increased	< 15

EDA = end diastolic area, ESA = end systolic area, FAC = fractional area change, CO = cardiac output, E/E' = ratio of transmitral peak E wave velocity to the early peak lateral mitral annular tissue velocity (E' = E_M = E "prime"), HD = hemodynamic.

1. Which of the following scenarios shown above would be expected with decreased systolic function following overzealous volume administration?
   A. Scenario A
   B. Scenario B
   C. Scenario C
   D. Scenario D
   E. Scenario E

2. Which of the following scenarios shown above would be expected with low systemic vascular resistance?
   A. Scenario A
   B. Scenario B
   C. Scenario C
   D. Scenario D
   E. Scenario E

3. Which of the following scenarios shown above would be expected with reduced left ventricular compliance?
   A. Scenario A
   B. Scenario B
   C. Scenario C
   D. Scenario D
   E. Scenario E

4. Which of the following scenarios shown above would be expected with hypovolemia (decreased intravascular volume status)?
   A. Scenario A
   B. Scenario B
   C. Scenario C
   D. Scenario D
   E. Scenario E

The correct findings are listed below:

HD Scenario:	Contractility	EDA	ESA	FAC	CO	E/E'
Hypovolemia	Normal	Decreased	Decreased	Normal	Decreased	< 15
Low LV compliance	Normal	Decreased	Decreased	Normal	Decreased	> 15
Decreased SVR	Normal	Normal	Decreased	Increased	Increased	< 15
Decreased Systolic function	Decreased	Increased	Increased	Decreased	Decreased	> 15
Nothing (trash)	Normal	Increased	Increased	Increased	Increased	< 15

EDA = end diastolic area, ESA = end systolic area, FAC = fractional area change, CO = cardiac output, E/E' = ratio of transmitral peak E wave velocity to the early peak lateral mitral annular tissue velocity (E' = E_M = E "prime"), HD = hemodynamic.

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NOTES

Unanticipated Ascending Aortic Dilation: How Big Is Too Big?

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The ascending aorta including the aortic root is the most common site for aneurysms of the thoracic aorta. It is estimated that approximately sixty percent of thoracic aortic aneurysms involve the ascending aorta or aortic root. The objectives for surgical operation to repair a dilated ascending aorta or aortic root include correcting associated aortic valve disease, eliminating the mass effect caused by the aneurysm, relieving refractory pain caused by the aneurysm, preventing aortic rupture, and preventing aortic dissection. In the absence of clinical symptoms, the decision to repair an ascending aortic aneurysm is based on weighing the risks associated with operation against the risks of eventual aortic rupture or dissection.

Clinical experience and epidemiologic studies of the natural history of thoracic aortic aneurysm suggested that aneurysm diameter was traditionally the most important factor predicting the risk of rupture or dissection. Based on the Yale University thoracic aortic aneurysm and dissection database, the cumulative lifetime risk of complications related to an ascending aortic aneurysm increased abruptly to 31% for patients with ascending aortic size $\geq$ 6.0 cm in diameter. This information, in combination with an estimated surgical mortality in the range of 2.5% to 5.0% for the repair of ascending aortic aneurysms, provided support for the general recommendation that surgical repair was warranted for patients with ascending aortic aneurysms of at least 5.5 cm in diameter.

Using aneurysm size greater than 5.5 cm as the only criteria for surgical repair to prevent complications of ascending aortic aneurysm has certain limitations. First, it has been recognized that measurement of aortic diameter may be associated with an uncertainty in the range of 0.5 cm caused by differences in imaging techniques or physician interpretation. Data used in analyzing the relation between aortic diameter and the risk of rupture or dissection were based primarily on axial cross sectional images obtained by computed tomography scans that could potentially overestimate the true cross sectional diameter of the aorta if an oblique view of the vessel was used for the measurement. Multiplane transesophageal echocardiography, intravascular ultrasound, or three-dimensional reconstruction of computed tomographic angiography studies may yield smaller aortic diameters because it is more likely that the true cross sectional diameter of the aorta can be measured even if the vessel is tortuous. Conventions applied to measure aneurysm diameter may also introduce variability among different imaging techniques. For example, the convention in CT angiography is to measure aneurysm diameter from vessel adventitia to adventitia, while the convention in ultrasound is to measure aneurysm diameter between the leading edges of the vessel wall. Second, aneurysm etiology has also been demonstrated to be an important risk factor predicting complication rates. For

this reason, surgical repair may be justified for ascending aneurysm diameter in the range of 4.0 cm to 5.0 cm in patients with Marfan syndrome, bicuspid aortic valve, or familial thoracic aortic aneurysm syndrome who exhibit higher rates of rupture or dissection at smaller aneurysm diameters. Finally, it is important to recognize that knowledge of the natural history of ascending aortic aneurysms is incomplete. Epidemiologic studies have analyzed and followed only a selected subset or limited number of patients without intervention until aneurysm rupture or dissection. Also, outcomes may have been influenced by reasons not to operate in certain patients with aneurysms, differences in criteria applied for surgical intervention, variations in the type of operations performed for repair, or the presence of co-morbid conditions that affect mortality.

It has been established that aortic aneurysm is a dynamic disease state and that aneurysms grow in size over time. For this reason, aneurysm growth rate is another important factor that influences the decision to repair a dilated ascending aorta. For example, knowing the aneurysm growth rate may be important for deciding whether to repair a dilated ascending aorta detected incidentally by intraoperative transesophageal echocardiography because delaying aneurysm repair until standard aneurysm size criteria have been satisfied would be associated with the added risks of re-operation. Data from the Yale Thoracic Aortic Aneurysm Database determined that the average rate of increase in diameter for ascending aortic aneurysms was 0.07 cm per year. However, reported growth rates for thoracic aortic aneurysm diameter in smaller series have ranged from 0.1 cm per year to as high as 0.42 cm per year. Factors associated with faster aneurysm growth rates were larger initial aneurysm diameter, the presence of dissection, Marfan syndrome, or poorly controlled hypertension. Anticipating eventual growth in the size of an ascending aortic aneurysm over time may be used to justify prophylactic replacement of the ascending aorta even if the aneurysm diameter is less than 5.5 cm at the time of aortic valve or root replacement operations in order to avoid the need for re-operation within 5 to 10 years. It is also possible to justify earlier surgical intervention in individual patients exhibiting rapid, sudden, or unpredictable increases in the size of their aneurysm over time.

Considerable controversy remains surrounding the questions of: how big is too big for the ascending aorta and when is surgical intervention warranted for repair of a dilated ascending aorta. Evolving surgical techniques and improved understanding of the natural history and pathophysiology of thoracic aortic aneurysms will likely refine criteria for surgical intervention. Existing evidence supports surgical intervention for ascending aortic aneurysms with a diameter $\geq$ 5.5 cm in all to prevent rupture or dissection. Operative repair of smaller ascending aortic aneurysms $\geq$ 5.0 cm may be justified in patients with aortic valve disease requiring aortic valve replacement. Repair of ascending aortic aneurysms as small as $\geq$ 4.5 cm in diameter may be justified in selected patients with conditions associated with an increased risk of eventual rupture or dissection such as family history of aortic dissection, Marfan syndrome, or bicuspid aortic valve if operative risk is acceptable.

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Echocardiographic Assessment of Myocardial Viability

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Introduction

Ischemic heart disease is a common cause of heart failure and is a leading cause of morbidity and mortality in developed nations. A central caveat of coronary revascularization is based on recovery of myocardial function after resumption of flow to a chronically underperfused heart in a region known as 'viable' myocardium. The concept of myocardial viability introduced by Rahimtoola in 1989 several years after a study reported that functional improvement was possible in ischemic myocardium.[1, 2] The term 'hibernating myocardium' was given to reversible wall motion defects in ischemic heart disease and encouraged coronary revascularization as a sound strategy. Essentially, the term refers to myocardium that is viable but exists in a state of contractile dysfunction due to hypoperfusion. Restoration of perfusion can reverse the hibernation and result in contractile recovery.

Restoration of perfusion, however, does not always lead to contractile recovery. A state of decreased contractility can exist despite adequate perfusion – known as myocardial stunning. A stunned myocardium is also viable, but has normal flow, in contrast to hibernating myocardium. It has been proposed that repetitive episodes of ischemia can induce myocardial stunning with normal resting perfusion. As coronary flow decreases over time, the stunned myocardium may also progress to hibernation. This progression may therefore represent a continuum in the evolution of ischemic heart disease.

Assessment of Hibernating Myocardium

Evaluation of myocardial viability is based on the contractile response to inotropic stimulation, identification of perfusion, or assessment of myocardial cellular and metabolic integrity. All these techniques involve administration of an 'uptake' agent and an imaging modality. Contractility can be enhanced by an inotrope such as dobutamine, while imaging can be performed with echocardiography (dobutamine stress echocardiography, or DSE), or magnetic resonance (MRI) scanning. Perfusion-based tests involve administration of intravenous contrast agents followed by echocardiography (myocardial contrast echocardiography, or MCE), MRI or CT scanning. These tests detect hypoperfusion and extent of scar tissue rather than contractile reserve. Metabolic tests assess cellular uptake of F^{18}-labelled glucose (FDG) or cell membrane and mitochondrial integrity with thallium or technetium–based radionulcides during nuclear imaging (PET or SPECT scanning)

However, with prolongation of the underperfused state, hibernating myocardium undergoes progressive loss of contractile elements (sarcomeres) that leads to contractile dysfunction. Basal metabolic functions, such as glucose uptake and cellular integrity remain intact. Therefore, while

contractility-based tests may not reveal viable tissue due to loss of contractile reserve, metabolic tests will demonstrate a 'normal' resting state. Consequently, contractility-based tests may be insensitive compared to perfusion/metabolic tests in the assessment of chronically hibernating myocardium. DSE is considered as a more specific test of viability, while thallium studies are least specific.

Echocardiographic Assessment of Myocardial Viability

Dobutamine Stress Echocardiogaphy (DSE)

Over the last 20 years, DSE has emerged as a popular, safe and cost-efficient technique for assessment of myocardial viability. Low dose dobutamine (5-10 mcg/kg/min) allows contractile reserve to be assessed. At this dose, viable myocardium demonstrates an improvement in contractility. Higher doses of dobutamine (up to 40 mcg/kg/min) are used to assess ischemia. The high dobutamine dose increases myocardial oxygen demand and if coronary flow is unable to meet the demand for increased perfusion, contractile function may worsen, indicating ischemia. Four different patterns of response to high dose DSE are recognized: (1) monophasic (initial improvement and no deterioration) suggesting viable myocardium with no coronary stenoses; (2) biphasic (initial improvement with subsequent deterioration) indicating viability with ischemia; (3) ischemic (deterioration without initial improvement) indicating severe ischemia with critical coronary stenosis; and (4) no change throughout study, representing a transmural scar. When DSE indicates viable myocardium as well as ischemia, functional response to revascularization is more likely.

Various studies have assessed the predictive power of DSE and it remains the most popular test of myocardial viability with a sensitivity of up to 86% and specificity of up to 94%. The wide range of these values is probably due to the use of different DSE protocols and non-randomized comparisons of techniques. Operator experience in interpretation of wall motion defects may also play a role. Higher volume centers may be able to detect viability with greater sensitivity and specificity.

There are a limited number of studies that have examined the value of intraoperative DSE in detection of viable myocardium either before or after revascularization. Seeberger and colleagues evaluated the contractile response to low-dose dobutamine in 80 patients undergoing coronary artery bypass surgery.[3] They concluded that DSE was a safe and sensitive technique for detecting inducible ischemia in patients with severe coronary artery disease and could be an important tool for cardiac risk stratification in anesthetized patients undergoing non-cardiac surgery.

Aronson et al assessed the value of a positive response to low dose DSE in predicting functional improvement after revascularization in 40 subjects undergoing CABG surgery.[4] Their study found that an improvement in wall motion after pre-bypass DSE was highly predictive of both, early and late improvement in wall motion after revascularization. Low dose intraoperative DSE was thus determined to be a sensitive indicator of contractile reserve and reliable predictor of functional recovery after revascularization.

Leung et al determined myocardial contractile reserve during reperfusion in the immediate post-bypass phase in 100 patients undergoing CABG surgery with low and high dose intraoperative DSE.[5] A subset of these cases had follow-up thallium perfusion studies after one week to evaluate myocardial viability. They found that while a monophasic response (see above) was highly specific for viable myocardium, while a biphasic or ischemic response was insensitive for

predicting non-viability. They suggested that new regional wall motion defects after revascularization can be subjected to DSE to discriminate between stunned myocardium and recurrent/persistent ischemia. If the defect shows improved contractility with DSE, stunning is the likely cause of the initial abnormality; while a biphasic response to high-dose DSE likely indicates recurrent ischemia and requires subsequent long-term follow-up.

In summary, DSE is a safe and efficient technique of assessing myocardial viability. It can provide useful information when used intraoperatively to evaluate the significance of new wall motion abnormalities in both, cardiac and non-cardiac surgery.

Myocardial Contrast Echocardiography (MCE)

Over the last decade, advances in microbubble technology have led to the development of intravenous contrast agents for use with echocardiography. Traditionally, 'agitated' saline has been used as intracardiac contrast due to the high echo reflective properties of gas in solution. The problem with gas bubbles is that they are highly unstable, and dissipate in solution due to the high surface tension and the fluid-gas interface and the pressure inside the spherical bubble. Microbubble perfusion agents have distinct characteristics that make them suitable for assessment of myocardial perfusion. These agents are essentially inert perflurocarbon gases surrounded by a lipid monolayer shell that impart surface stability. These agents are small enough (2-4 microns) to pass through the pulmonary circulation and can therefore be given intravenously; and follow rheological principles similar to red blood cells and can therefore enter the microcirculation. Microbubbles osscilate and can be expanded or fragmented by ultrasound waves. The individual bubbles will resonate depending on applied frequency and will reflect high quality signals that can be used to detect their presence in the microvascular bed. The problem is that they are also destroyed by high frequency ultrasound at high power.

The quantification of myocardial perfusion depends on the fragmentation of microbubbles by ultrasound and their replenishment by intact microbubbles, which depends on myocardial blood flow. The rate at which the acoustic intensity from the myocardium increases reflects myocardial blood flow velocity. This rate will plateau at some point when steady-state is achieved. The acoustic intensity at steady state reflects myocardial blood volume, since all of the microbubbles are intravascular. Also, about 90% of myocardial blood volume is contained in the capillary bed, which makes steady state acoustic intensity a reliable marker of capillary blood volume. Perfusion defects in the myocardium readily show up as filling defects on MCE. Since myocyte loss is accompanied by loss of microvasculature, MCE is an excellent tool for assessing both, myocardial microvascular perfusion and viability.

During MCE, segments that show normal or patchy perfusion are classified as viable, while those that do not show perfusion are deemed non-viable. Infarcted segments with adequate contrast perfusion have a better clinical outcome compared to segments with a perfusion defect.[6] Studies have shown that MCE is a very sensitive method for detecting myocardial viability, but its inability to detect subendocardial necrosis makes it less specific in predicting functional recovery after revascularization.[7]

Few studies have examined the role of MCE with regard to viability in the operating room. Intraoperative MCE for assessment of perfusion after revascularization was reported by Kabas et al in 1990.[8] They however, had used intracoronary injections of contrast agents to assess perfusion. Aronson and colleagues reported that myocardial segments that showed adequate perfusion predicted function at 6 weeks and one month after coronary bypass surgery.[9] Their

study suggested that MCE was a good technique for identifying myocardial viability after revascularization and for predicting postoperative systolic function. Another area this technology has been used intraoperatively has been in the assessment of distribution of retrograde cardioplegia. However, over the years, intraoperative MCE has not lived up to its expectations, despite great success with transthoracic imaging. A significant limitation of this technology has been that the higher frequency and mechanical indices used with TEE lead to greater rates of microbubble fragmentation. Moreover, myocardial segments in the far field are difficult to image due to higher 'dropout' rates as a result of acoustic shadowing. This may lead to segments being mislabeled as a perfusion defect or not labeled at all despite having a perfusion defect. Further refinements in transducer technology may enable the use of MCE as an efficient technique for assessment of myocardial viability.

Tissue Velocity Imaging

While most echocardiographic viability techniques focus on systolic thickening, the most sensitive indicators of ischemia are diastolic function parameters. Diastolic abnormalities are not only early indicators of ischemia, but tend to persist longer than systolic abnormalities. Tissue Doppler techniques allow accurate quantification of diastolic myocardial velocities and are sensitive to changes in myocardial perfusion. Abnormalities in diastolic myocardial velocities accurately differentiate between perfused and scarred tissue when used in conjunction with DSE.[10] It has also been reported as a more sensitive predictor of perfusion anomaly compared to DSE alone.[10] However, tissue Doppler is limited by angle of interrogation and translational motion of the heart. Strain and strain rate imaging (SRI) avoids some of this limitations and may be more accurate for identifying diastolic abnormalities. Strain refers to the amount of deformation between two echogenic points in the myocardium. Strain rate refers to the rate of this deformation. When using tissue Doppler, SRI can be performed on two points along the ultrasound beam. The advent of two-dimensional strain analysis by speckle tracking allows angle-independent assessment of strain, or 2D strain. Hoffman et al showed that when SRI with tissue Doppler was used with DSE, it identified subtle early diastolic abnormalities in non-viable segments.[11] There is limited literature with regard to speckle tracking and myocardial viability. Studies are underway to determine the feasibility of 2D strain and SRI in not only predicting systolic and diastolic function, but also myocardial viability in response to inotropic stimulation.

Future Directions

The oblique spiral fibers of the heart impart a wringing motion at the base, also known as LV torsion. The systolic torsion is then followed by an 'untwisting' or recoil of torsion that occurs during the isovolumic relaxation time. During this early diastolic phase, the 'untwist' contributes to the diastolic suction effect that leads to rapid early LV filling. When seen from the apex, LV rotation is described as clockwise at the base and counterclockwise at the apex. Two values are considered important in the measurement of rotation – degree of rotation and rotational velocity. The reversal of both values in diastole (negative twist) represent the 'untwist' parameters that describe early LV diastolic function. Both, degree and velocity of negative twist are delayed in patients with LV hypertrophy and may be early indicators of diastolic relaxation abnormality.[12] This modality may also help provide further insight into subtle changes in diastolic function that are seen in the immediate postoperative period. Further studies of twist and untwist may help with more accurate quantification of myocardial viability.

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Echo for Adult Congenital Heart Disease

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The number of adults with congenital heart disease (ACHD) has increased due to significant advances in diagnosis, medical and surgical care over the past five decades; at the moment in the United States the population of patients with ACHD is approaching one million. (1). The incidence of in the United States is estimated to be 6.2 per 1000 live births. Approximately 85% of these infants are expected to survive to adulthood (2). Survival of patients with ACHD rates depend on mutiple factors; year of birth, age at diagnosis, complexity of the lesion and whether or not a lesion has been surgically corrected or palliated (Table 1) (2). Definitive surgical repair at a very early age and the improved care of patients with complex CHD will result in improved survival in the future.

Congenital heart disease can be classified in several ways:

A) Simple lesions include isolated lesions such as intracardiac communications; complex pathology includes severe malformations or malpositions of the cardiac structures.

B) Acyanotic versus cyanotic lesions: Cyanotic conditions are usually associated with restrictive pulmonary blood flow in the presence of intracardiac shunting or complete arterial and venous admixtures.

C) Shunt, obstructive, regurgitant and mixed lesions: This classification is based on the physiologic spectrum of CHD. Shunt lesions may occur at the intracardiac or extracardiac levels. The direction and magnitude of the shunt depends on the size of the communication and the relative resistances of the pulmonary and systemic circulation. Shunts that are primarily left to right result in pulmonary overcirculation, ventricular volume overload and signs and symptoms of congestion. Right to left shunts due to pulmonary outflow tract obstruction or elevated pulmonary vascular resistance are seen in with hypoxemia. Obstructive lesions, of varying severity, may affect inflow or outflow regions of the heart. Regurgitant lesions rarely occur in isolation. Mixed lesions have complete mixing of the pulmonary and systemic venous return and circulation that exist in a parallel rather than in series arrangement.

Atrial Septal Defects

Atrial septal defects (ASD) account for one quarter to one third of all congenital lesions, occurring more commonly in women (3). There are four types of atrial septal defects:

(1) Ostium secundum defect (75%) which occurs in the mid septum region of the fossa ovalis. Varying degrees of mitral valve prolapse and mitral regurgitation can occur.

(2) Ostium primum defect (15%) which is also a form of a partial atrioventricular canal defect and consists of an defect in the lower aspect of the interatrial septum. Abnormalities of the atrioventricular valve can occur with a "cleft" in the anterior mitral valve and septal tricuspid valve leaflet with variable degrees of regurgitation.

(3) Sinus venosus defect (10%) is usually superior and posterior in relation to the superior vena cava (more frequently) and/or inferior vena cava. These defects are commonly associated with an anomalous drainage of one or more pulmonary veins into the right atrium or superior vena cava (4).

(4) Coronary sinus defects (extremely rare) occur between the left atrium and coronary sinus and can be associated with a persistent left superior vena cava.

A defect in the interatrial septum allows pulmonary venous blood to pass from the left to the right atrium. A patient with a large defect can develop atrial enlargement, dilation of the right ventricle and pulmonary arteries and develop pulmonary hypertension. The potential detrimental effects of chronic right ventricular volume overload, paradoxical embolization and atrial dysrhythmias are considered indications for intervention.

Primary or patch closure of an atrial septal defect in childhood provides excellent operative results and nearly normal long-term survival in adults (5,6). Atrial arrthythmias may be seen especially after the third decade of life. Operative patch closure is generally recommended if the shunt is large with a pulmonary blood flow–to–systemic blood flow ratio of 1.8:1 or greater and right ventricular enlargement. In some cases with favorable anatomy, closure of the secundum defects may be accomplished by the transcatheter route (7).

Intraoperative TEE is performed to confirm the presence, size and location of the defect, degree of atrioventricular valve regurgitation, ventricular function and to evaluate for associated anomalies such as anomalous pulmonary veins, systemic venous obstruction or erroneous diversion of systemic venous drainage to the left atrium. Color flow Doppler echocardiography can demonstrate flow across the atrial septum and detect mitral or tricuspid regurgitation. To evaluate for small shunts agitated saline (contrast) can be used. Postoperatively, TEE is used to detect residual shunts by color Doppler and contrast, assess ventricular function and assess for pulmonary venous obstruction and valvular regurgitation.

Ventricular Septal Defects

Ventricular septal defects (VSDs) are the commonest cardiac abnormality in infants and children (8). Adults with unoperated VSDs are encountered less frequently than those with ASDs because large defects are usually closed surgically in childhood when there is evidence of congestive heart failure or pulmonary hypertension. Also, in infancy and childhood, defects have a greater rate of spontaneous closure (90% of those that close do so by the time the child is 10 years of age) (9). Small perimembraneous and trabecular VSDs may completely close spontaneously even in adulthood (9).

Ventricular septal defects can be classified by anatomic location into four types

(1) Perimembranous defects (70%) are found in the membranous region of the septum and can extend into the muscular, inlet or outlet regions.

(2) Muscular defects (20%) are located within the trabecular portion of the septum, and can be in the central or apical areas. Multiple defects can occur, either two or three or multiple small ones known as "swiss cheese septum".

(3) Doubly committed or subarterial ventricular septal defects – so called "supracristal" defects (5%) are found just below the aortic and pulmonary valves and may have associated aortic cusp herniation and aortic regurgitation.

(4) Inlet ventricular septal defects (5%) occur close to the atrioventricular valves in the posterior portion of the septum.

These septal defects permit left-to-right shunting at the ventricular level and the physiologic consequences are determined by the size of the defect and the relative resistances of the systemic and pulmonary vascular beds. If the defect is restrictive, as described above, there is a large pressure gradient between the ventricles in systole and the flow across the ventricular septal defect is limited. If the defect is large (nonrestrictive), there is minimal to no pressure difference between the left and the right ventricles and the magnitude of the shunt is dependent upon the ratio between the pulmonary and systemic vascular resistance.

In adults with VSD, the overall 10-year survival after initial presentation is approximately 75%. Patients who survive to adulthood comprise two groups, those whose VSD's have closed spontaneously or those with nonrestrictive defects that can develop elevated pulmonary vascular resistance. About 10% of patients with nonrestrictive VSD's develop Eisenmengers (9). syndrome or reaction, which is defined as nonrestrictive VSD, pulmonary vascular disease and reversed shunt.

TEE is particularly helpful in evaluation of these defect. TEE should be used to assess the location and size of the defect, to determine the chamber sizes and PA dimensions, to detect associated anomalies, to identify ventricular septal aneurysms if present, to evaluate the aortic valve for herniation, prolapse or insufficiency, and to determine if there is pulmonary hypertension. Color flow Doppler can be used to identify the direction and magnitude of shunt flow, and the presence of any associated valvular regurgitation. Pulsed and continuous wave Doppler can be used to determine the peak velocity across the VSD. When restrictive, the velocity is high whereas when nonrestrictive the velocity would be low. The peak velocity is then used to calculate the right ventricular systolic pressure (RVSP) which is equal to the pulmonary artery systolic pressure (PASP) in the absence of any outflow obstruction.

The formula used is RVSP=SBP-(peak velocity of VSD jet)2 (10). Postoperatively, TEE is used to detect residual shunts by color Doppler and contrast, assess ventricular function and residual aortic or pulmonic insufficiency and right ventricular outflow tract obstruction.

Complete AV Septal Defect

In complete AV septal defect (AVSD), or atrioventricular canal defects, there is failure of the endocardial cushions to close the atrial and ventricular septum, affecting the complete formation of the mitral and tricuspid valves. As a result, patients have a ventricular septal defect, an atrial septal defect, and varying degrees of AV valve regurgitation. AVSD are calssified into several forms:

Partial: where the ventricular septum is intact , almost always a primum ASD and cleft in the mitral valve

Intermediate: the rarest from with a restrictive VSD, primum ASD and cleft mitral valve

Complete:Nonrestrictive VSD, primum ASD and a common orifice for the atrioventricular valves.

With large left-to-right shunts at the atrial and ventricular levels and systemic pressures in the right ventricle, pulmonary vascular disease occurs very early. Most adults with uncorrected complete atrioventricular septal defects (AVSD) are cyanotic with Eisenmenger's syndrome and are not candidates for correction.The lesions in these patients

are usually corrected in infancy. The surgery consists of patch closure of the atrial and ventricular septal defects and repair of the AV valves. The unoperated adult with a complete AV septal defect is rarely a candidate for a complete repair because of the development of pulmonary vascular disease, but they may be a candidate for heart-lung transplantation or lung transplantation with intracardiac repair.

TEE is useful for defining the type and extension of the septal defects (Table 2) and the morphology of the "bridging leaflets", which span the common orifice. Proposed by Rastelli et al. (11), the attachment of the anterosuperior leaflet defines the various types of AVSD's (a, b and c). TEE of an AVSD demonstrates complete absence of the crux of the heart, with both low atrial and high ventricular septal defects. In the adult, color-flow imaging and Doppler studies usually show regurgitation of both AV valves and evidence of pulmonary artery hypertension.

Left ventricular outflow tract obstruction

In congenital left ventricular outflow tract obstruction (LVOTO), the anatomic level of obstruction can be supravalvular or subvalvular whereas in acquired aortic stenosis, it is almost always valvular. Valvular LVOTO in the adult patient is usually due to bicuspid aortic stenosis, occurring due to fusion of the cusp resulting in a raphe or false commisure.. It occurs in 2-3% of the population, usually occurring in isolation but can be associated with other anomalies, the most common of which is coarctation of the aorta, PDA or ascending aortopathy. Patients with the most severely malformed and stenotic valves may require intervention in childhood. In supravalvular aortic stenosis, the narrowing is usually above the level of the sinuses of Valsalva. The coronary arteries arise proximal to the area of obstruction and are subjected to elevated systolic pressures equal to that of the left ventricle. This may lead to coronary artery dilatation and accelerated atherosclerosis.

In the most common form of subaortic stenosis, there is a discrete membrane or fibromuscular ridge immediately underneath the aortic valve that either partially or completely encircles the let ventricular outflow tract or a fibromuscular narrowing of the whole left ventricular outflow tract with a small aortic root. There is association of left ventricular outflow tract obstruction with coarctation of the aorta, mitral valve abnormalities (parachute mitral valve , supravalvar mitral ring) which is known as Shone's syndrome.

Aortic stenosis occurs in about 5% of all CHD lesions and bicuspid valves are most common in patients with symptomatic AS. Patients may remain asymptomatic for years but are at risk for developing endocarditis, AS and AR. Approximately one-fourth of patients requiring surgical intervention in childhood will need repeat surgery for recurrent stenosis or progressive aortic insufficiency in the next 25 years (12). In the adolescent and adult, a variety of surgical approaches are available for the management of valvar aortic stenosis. TEE evaluation should include determination of the aortic valve morphology, measurement of annulus size, identification of the post stenotic dilatation of the ascending aorta, delineation of a subaortic membrane and estimation of the pressure gradient across the left ventricular outflow tract by Doppler interrogation. Aortic coarctation, pathology characteristic of Shone's complex, left ventricular hypertrophy and function should also be assessed. Color flow Doppler can be used to assess the aortic regurgitation after valvuloplasty, surgical valvotomy or aortic valve replacement. Pulsed and continuous wave Doppler can be used to evaluate gradients across the LVOT.

Coarctation of the Aorta

In the most common form of aortic coarctation, there is narrowing immediately distal to the take off of the left subclavian artery. The constriction may take the form of a shelf or a localized hourglass narrowing of the distal arch proximal to the ligamentum arteriosus.

Arterial hypertension is usually present proximal to the aortic obstruction. Repair is usually undertaken when the coarctation is severe enough to cause proximal hypertension and the gradient across the coarctation is greater than 25 to 30 mm Hg. Surgery can be complicated by the presence of multiple collateral vessels (suggested on chest radiograph by notching) and atheromatous changes at the site of the coarctation. In some patients, primary balloon angioplasty with or without stenting has been used successfully. Aortic aneurysms can occur around the area of coarctation or elsewhere in the aorta and in the branches of the circle of Willis (so-called berry aneurysms). Essential hypertension is a common finding in adult.

It is difficult to visualize the actual site of the coarctation with TEE. However, color Doppler interrogation of the descending aorta may detect flow acceleration, turbulent jets (13) or a pressure gradient, which continues into diastole (14). An associated bicuspid aortic valve can also be identified by TEE. Angiography or MRI is usually needed to define the site and extent of narrowing.

Pulmonary Valve Stenosis

Although pulmonic valve stenosis is congenital in origin it may be progressive. The obstruction to outflow adds an afterload burden on the right ventricle, resulting in right ventricular hypertrophy. Severe right ventricular hypertrophy with increased systolic compression may compromise intramural coronary flow.

It is unusual to see an adult with severe valvular pulmonary stenosis. Adult patients with valvular pulmonary stenosis usually do well, but eventually right ventricular failure can occur. Surgical valvotomy has been an extremely successful operation for long-term relief of pulmonary valve obstruction. In patients with severe valvular pulmonary stenosis, percutaneous balloon valvuloplasty is the current treatment of choice and has replaced surgical valvotomy in patients with flexible valves (15-17).

TEE demonstrates the stenotic doming pulmonary valve and thickening of the free wall of the right ventricle. The orifice can range from a pinhole to several millimeters but is rarely critical in the adult. The pulmonary valve is best seen in the basal horizontal views where it is seen in long axis (ME asc aortic SAX and RV inflow-outflow view). Accurate measurements of the pressure gradient across the right ventricular outflow tract are possible with Doppler interrogation, usually from the transgastric view.

Patent Ductus Arteriosus

The patent ductus arteriosus, which account for 10% of cases of congenital heart disease, connects the proximal descending aorta with the pulmonary artery at its bifurcation. The magnitude of the left-to-right shunt depends on the size of the ductus and the ratio between the pulmonary and systemic vascular resistances in a manner similar to that of a ventricular septal defect.

The marked increase in pulmonary blood flow results in left-sided volume overload with dilation of the left-sided cardiac structures. The left ventricular volume overload can result in congestive heart failure. Similar to a large VSD, the long standing high pressure, high flow state can cause severe pulmonary hypertension and Eisenmenger's syndrome as a result of pulmonary vascular disease. Currently, some centers use a video-assisted thorascopic approach for ductal closure with excellent results (18). Percutaneous closure using coils or intravascular devices is feasible in some patients with an approximately 95% success rate at intermediate follow-up (19); long-term follow-up is not yet available.

On two-dimensional echocardiography, the ductus itself is rarely seen, but visualization of abnormal, continuous, high-velocity "aliased" flow within the main pulmonary artery near the left branch is seen on color-flow Doppler imaging. Elevation of PA pressures can be diagnosed either by continuous wave Doppler of the tricuspid and/or pulmonary regurgitant jet(s) or by continuous wave Doppler of the PDA flow. Systolic PA pressure can be calculated by using the formula: 4 x peak PDA flow^2 minus systolic arterial pressure. Diastolic PA pressure can be calculated by using: 4 x PDA flow at end diastole^2 minus diastolic arterial pressure.

Coronary Artery Anomalies

Myocardial ischemia secondary to congenital coronary artery anomalies is a rare cause of exertional syncope or chest pain but should be considered if these symptoms occur in young patients (usually in the second or third decade of life). The most common coronary anomalies seen in adults are anomalous origin of the left circumflex coronary artery from the right sinus of Valsalva, coronary to pulmonary artery fistulas, coronary cameral fistulas (fistulous connection between the coronary artery and a cardiac chamber, usually right atrium or right ventricle), and abnormal origin of the left coronary artery from the anterior sinus of Valsalva or the right coronary artery from the left posterior sinus of Valsalva. TEE may be used for monitoring of ventricular function and ischemia (21).

Tetralogy of Fallot

Tetralogy of Fallot, the most common cyanotic defect after infancy refers to a combination of lesions consisting of a) an interventricular septal defect, b) right ventricular outflow tract obstruction c) an aorta overriding the ventricular septal defect d) right ventricular hypertrophy, which is a compensatory response to the pressure load. The right ventricular obstruction and unrestricted, large ventricular septal defect result in an elevated right ventricular pressure that is similar to systemic pressure.

Most patients with tetralogy of Fallot have had palliative operations or corrective surgery by the time they are teenagers. Occasionally, a patient reaches the third decade of life without surgery. Sometimes patients present with only palliative systemic to pulmonary arterial shunts such as Blalock-Taussig shunt (subclavian artery to pulmonary artery), Potts shunt (descending aorta to left pulmonary artery), or Waterston shunt (ascending aorta to right pulmonary artery), central interposition tube graft, infundibular resection (Block procedure) or pulmonary valvotomy, right ventricular to pulmonary artery conduit without VSD closure or with fenestrated closure. Prior to surgical correction, most patients died in the second decade of life.

Although it is an extremely successful operation, complete repair of tetralogy of Fallot has several potential significant postoperative residua, including residual right ventricular outflow tract obstruction, pulmonary regurgitation, peripheral pulmonary artery stenosis of one or both pulmonary arteries and ventricular septal patch leaks. Survival in postoperative tetralogy of Fallot is about 90% at about 30 years after surgery.(22). Thus, most adults with tetralogy of

Fallot come to surgery for correction of significant hemodynamic sequelae, especially pulmonary outflow pathology and less frequently for residual VSD.

TEE is useful for definition of the pathology, which is best seen in the mid-esophageal long axis view . In patients with unrepaired tetralogy of Fallot, TEE demonstratesvarious degrees of right ventricular outflow obstruction at the infundibular, valvar, supravalvar and branched levels. There is a large ventricular septal defect in the vicinity of the membranous septum with evidence of right-to-left shunting and a dilated overriding aorta. The gradient across the right ventricular outflow tract can be measured with spectral Doppler by interrogating the main pulmonary artery in a mid esophageal ascending short axis view of the aorta (21).

Truncus Arteriosus

In truncus arteriosus, the embryonic truncus fails to divide into an aorta and a pulmonary artery, retaining the single semilunar valve. A ventricular septal defect is almost always present. The pulmonary, aortic and coronary arteries arise from the single truncal root. Classification is based on the anatomic origin of the pulmonary arteries from the single trunk (23,24). Either the pulmonary trunk arises separately from the common trunk and close to the truncal valve (Type I), or as separate vessels from the truncus (Type II and III).

The truncus patient is usually managed surgically by closing the VSD and incorporating the semilunar valve on the left side as the aortic valve. A valved conduit is placed from the RV to the pulmonary artery. Depending on the severity of the pulmonary artery stenoses varying techniques including surgical and balloon catheter techniques are used to relieve the obstruction. Late complications that constitute indications for surgery in the adult patient with prior truncus repair include pulmonary outflow conduit failure resulting in either obstruction or regurgitation, residual obstruction to the distal pulmonary arteries, and truncal valve regurgitation.

In the unoperated patient, TEE demonstrates the presence of a single semilunar valve, the size and location of the VSD and provide evidence of left ventricular volume. If the pulmonary arteries arise without stenosis from the truncus and the VSD is large, pulmonary blood flow will be markedly increased, volume loading the LV and resulting in congestive symptoms and pulmonary hypertension. Without surgical intervention, patients do not usually survive to adulthood.

Transposition of the Great Arteries

These infants are born with great arteries arising from the inappropriate ventricle. The aortic valve arises anteriorly from the right ventricle and the pulmonic valve posteriorly from the left ventricle. Without mixing at the atrial or ventricular levels or patent ductus arteriosus, this lesion is incompatible with life.

Infants with this condition rarely survive without intervention. With only rare exceptions, adults have had a palliative atrial switch procedure (such as the Mustard or Senning operation), after which the right ventricle usually continues to serve the systemic circulation. One current preferred surgical approach is the arterial switch operation (Jatene procedure) designed to reconnect the great arteries to their proper ventricle, translocate the coronary arteries to the new aortic location, and restore a normal physiology.

TEE evaluation with multiple planes including contrast injection should include baffle assessment after the Senning and Mustard procedure to evaluate potential baffle leaks or venous obstruction. Evaluation of ventricular function is important as patients with an atrial switch procedure are at risk of right ventricular (systemic pump) failure. Patients with the arterial switch operation (Jatene procedure) are at risk for coronary artery obstruction, atrial or ventricular patch leaks, aortic regurgitation, and supravalvular obstruction at the site of the aortic and pulmonary artery anastomoses.

Physiologically "Corrected" Transposition of the Great Vessels (l-TGV)

This lesion is characterized by malposition of the great vessels and ventricular inversion, with the left ventricle connected to the right atrium and pulmonary artery and the right ventricle connected to the left atrium and aorta. Blood flow is physiologically correct, but an anatomic right ventricle serves as the systemic ventricle. Most previously undiagnosed adults with this lesion usually have no other abnormalities. Corrected transposition is frequently discovered as a result of the physical findings due to accompanying congenital defects such as VSD, subpulmonic valve stenosis or tricuspid regurgitation. With no other abnormalities, this lesion may remain undetected until the patient presents with syncope owing to complete heart block.

Without associated lesions, patients with corrected transposition may live many years. The development of complete heart block is common and occurs at a rate of approximately 5% per year in adults. Surgical correction may be possible (atrial plus arterial switch) but might not impact mortality compared to conservative management (25). The systemic ventricle, an anatomic right ventricle, may show progressive dysfunction requiring medical therapy and resulting in tricuspid regurgitation from annular dilation. Adults with l-TGV rarely require surgery. Patients previously operated upon for pulmonary obstruction may have conduit stenosis or valve failure.

TEE examination should include identification of ventricular morphology and function, associated lesions such as VSD and pulmonary outflow tract obstruction.

Ebstein's Anomaly

In Ebstein's anomaly, the septal and posterior leaflets of the tricuspid valve are dysplastic and displaced from the tricuspid annulus apically into the body of the right ventricle (26-28). Thus, a portion of the right ventricle lies above the tricuspid valve (the atrialized right ventricle) and enlarges the true right atrium. The tricuspid valve may be regurgitant, and if a patent foramen ovale or an atrial septal defect is present (80% patients), there can be a large right-to-left shunt. These patients are cyanotic, are likely to present in infancy, and may have had palliative procedures with closure of atrial level shunts. The presentation in adolescents or adults is more likely related to progressive tricuspid regurgitation and heart failure or arrhythmias than to cyanosis. Nevertheless, some patients with severe tricuspid valve displacement can live normal lives with minimal symptomatology.

Once the patient has survived childhood, the prognosis is favorable, even with severe forms of Ebstein's anomaly. If tricuspid regurgitation is severe, the ability to increase cardiac output may be diminished and the patient may have progressive exercise limitation with fatigability. Closure of an atrial septal defect and reconstruction of the tricuspid valve or even replacement has been done with success. If atrial tachycardia is present with an anomalous atrioventricular conduction pathway (Wolff-Parkinson-White syndrome), radiofrequency ablation of the accessory connection can successfully treat the paroxysmal atrial tachycardia.

The TEE is diagnostic, using the mid-esophageal 4 chamber - and right ventricular inflow-outflow view. The abnormal tricuspid valve has a large, redundant anterior leaflet and displaced, immobile septal and posterior leaflets . The tricuspid regurgitant jet arising apically within the right ventricle and the right atrium and ventricle are enlarged.

Single Ventricle

A number of structural cardiac lesions are included in the single ventricle (univentricular heart) category. Hypoplasia of either the RV or LV results in a functional single ventricle. Lesions in this group include hypoplastic left heart syndrome, double inlet left ventricle and tricuspid atresia. Other cardiac malformations with two ventricular chambers may also be considered in the functional single ventricle group because of associated defects that preclude a biventricular repair. Adult patients with single ventricle lesions may have palliative procedures such as: a modified Blalock Taussig shunt, a Glenn anastomosis (superior vena cava to pulmonary artery cavopulmonary connection) (29) or a Fontan connection (total caval to pulmonary connection) (30,31). The Fontan procedure includes a variety of surgical modifications, which connect either the right atrium or the venae cavae directly to the pulmonary arteries, bypassing the RV. Pulmonary blood flow is passive and dependent on the transpulmonary pressure gradient as well as a low pulmonary vascular resistance (PVR).

The Fontan operation is a palliative procedure for patients with a functionally or anatomically single ventricle or complex malformation considered unsuitable for biventricular repair. There is diversion of all of the systemic venous return to the pulmonary arteries, usually without employing a subpulmonary ventricle.
Originally described for patients with tricuspid atresia, it has now been extended to most forms of single ventricle circulation.

There are numerous variations in the surgical approach. The most likely types of Fontan procedure to be encountered today are: 1) the direct RA-PA connection; 2) the total cavopulmonary connection (SVC to PA and IVC to PA through an intra-atrial tunnel); 3) the extra cardiac conduit (SVC to PA and IVC to PA through an external conduit); and 4) RA-RV through a valved conduit when RV size and function is adequate. The Fontan procedure may be done as a single or staged procedure (with a classic or bi-directional Glenn shunt performed as the first procedure, followed by the completion of the Fontan as a second procedure). The Glenn procedure is usually performed in early childhood. Progressive desaturation as well as polycythemia and potential benefits of separation of the circulation are considered indications for a Fontan procedure. This surgical intervention is usually performed during childhood but occasional adults present for the procedure or require revision of a prioe surgery. Patients with the Fontan procedure can do well into the 3rd and 4th decade. Patients may have elevated systemic venous pressures and may develop ascites and peripheral edema monthsor years following surgery. Other long term complications include protein losing enteropathy and ventricular dysfunction. Atrial brady-tachyarrythmias, especially in those patients where the right atrium is incorporated into the connection to the pulmonary artery, can be a challenging problem. Adult patients with single ventricle physiology may come to surgery for revision of prior palliative or "definitive" procedures, or transplantation.
TEE evaluation should include caridac assessment of atrioventricular, ventriculoarterial connections and ventricular size and morphology . Visualization of the Glenn anastomosis is not always possible by TEE but it can sometimes be demonstrated using mid esophageal bicaval views. TEE is particularly useful for evaluation of ventricular function, valvular regurgitation, and for excluding thrombus or known pathway obstruction.

Conclusions

Adults with CHD may come to the attention of anesthesiologists during:

a) cardiac surgery for the first time, for either definitive repair or palliation
b) cardiac surgery for definitive correction following palliative surgery
c) cardiac surgery for management of complications of prior intervention, or conversion of a repair to a more modern technique
d) non cardiac surgery in the presence of untreated, palliated, or corrected lesions.

In many medical centers, TEE is now used for intraoperative assessment of patients with congenital heart disease undergoing cardiac surgery. In non cardiac surgery, where additional monitoring may be required,TEE can be used to evaluate ventricular volume, function, underlying pathology and assess any residual lesions.

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You're the Intraoperative Echocardiography Consultant

Moderators: GS Hartman/SK Shernan

6:45 – 7:30 am Continental Breakfast

7:30 – 8:30 am **Review of Advanced Cases**
GS Hartman, SK Shernan

8:30 – 10:30 am **You're the Intraoperative Echocardiography Consultant**
Faculty: K Glas, AD Maslow, CG Koch, GS Hartman, SK Shernan

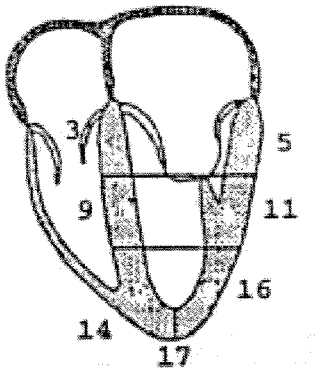
NOTES

You're the Intraoperative Echocardiography Consultant: Case Discussions with the experts

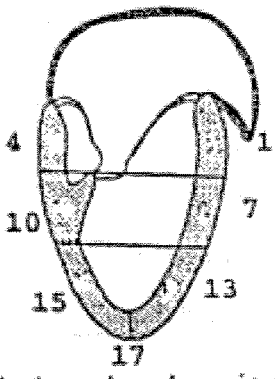
Drs. Hartman, Shernan, Glas, Maslow, Koch

During this session, you will be asked to review a series of TEE examinations and provide your "read" or interpretation. Following these reviews, the panelist will then "read" their studies aloud, highlighting the important findings. An opportunity for questions and answers will follow each selected study.

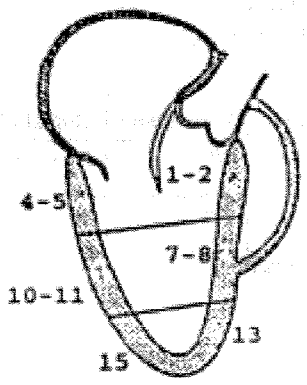
The SCA sample perioperative echo report template is provided on the following pages to facilitate and organize your interpretations.



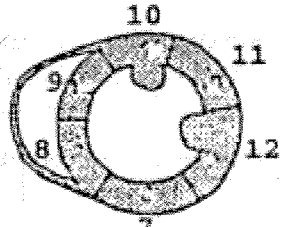
a. four chamber view



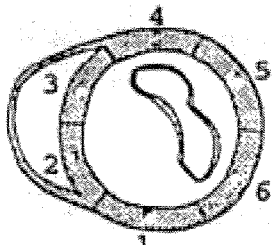
b. two chamber view



c. long axis view



d. mid short axis view



e. basal short axis view

1. basal anterior
2. basal anteroseptal
3. basal inferoseptal
4. basal inferior
5. basal inferolateral
6. basal anterolateral
7. mid anterior
8. mid anteroseptal
9. mid inferoseptal
10. mid inferior
11. mid inferolateral
12. mid anterolateral
13. apical anterior
14. apical septal
15. apical inferior
16. apical lateral
17. apex

Sample Perioperative Echo Report

1. Demographic and Other Identifying Information

Name_____ Unit #_____DOB_____ M/F Date_____ Time____
Location: ICU/OR/ER/PACU, Phys requesting Echo _____, Examiner_____
Indication_____ CPT Codes_____ICD-9 codes_____
Intubated: Y/N, Sedated: Y/N, Insertion: Easy/Difficult /Failed Probe Type: pediatric/ biplane/multiplane/ epicardial/epiaortic Modalities: 2D/CFM/PWD/CWD/Contrast/Pharm/Stress Tape/OD #_____

2.Echocardiographic and Doppler Measurements

Aorta	Size	Diam (cm)	Dissection	Plaque thick (mm)	Plaque mobile
Ascending Ao	nl, dilated, aneur		y/n	0-3, >3	y/n
Ao Arch	nl, dilated, aneur		y/n	0-3, >3	y/n
Descending Ao	nl, dilated, aneur		y/n	0-3, >3	y/n

Valves	Annulus	Stenosis	Area/ Gradient	Regurg	Leaflet Morphology	Leaflet Motion
Aortic Valve	normal, dilated, calc, bioprosth, mechanical	No, mild, Mod, severe		0, 1+, 2+, 3+ 4+	Nl, Calc, Veg, Perf, Bicuspid Thickened,	Nl, Prolapse Flail, Restricted NCC/RCC/LCC
Mitral Valve	normal, dilated, calc, bioprosth, mechanical	No, mild, Mod, severe		0, 1+, 2+, 3+ 4+	Nl, Calc, Veg Perf, Myxom Thickened	Nl, Prolapse, Flail, SAM, Restricted A_1,A_2,A_3/ P_1,P_2,P_3
Tricuspid	normal, dilated, calc, bioprosth, mechanical	No, mild, Mod, severe		0, 1+, 2+, 3+ 4+	Nl, Calc, Veg Perf, Myxom Thickened	Nl, Prolapse Flail, Restricted

Atria	Size	SEC (smoke)	Thrombus	Tumor	Device
Right atrium	normal, dilated	y/n	y/n	y/n	y/n
Left atrium	normal, dilated	y/n	y/n	y/n	y/n

Left atrial appendage:

Interatrial Septum: Morphology: Normal, Aneurysm Lipomatous Hypertrophy, PFO, ASD (Primum, Secundum, Sinus Venosus, AV Canal) Shunt: (r->l, l->r, bidirectional)

Interventricular Septum Morphology: Normal, Hypertrophy, Shift, Defect (membr, musc), Shunt: (r->l, l->r)

Ventricles	Cavity size	Cavity Dimension	Hypertrophy	Thrombus	Global FXN	EF
RV	normal, dilated		y/n	y/n	Nl, ↓, ↓↓,↓↓↓	
LV	normal, dilated		y/n	y/n	Nl, ↓, ↓↓,↓↓↓	

Regional Function: (1=normal, 2= hypokinetic, 3= akinetic, 4=dyskinetic)

Basal Segments	Mid Segments	Apical Segments
Anterior	Anterior	Anterior
Anteroseptal	Anteroseptal	Septal
Inferoseptal	Inferoseptal	Inferior
Inferior	Inferior	ateral
Inferolateral	Inferolateral	True apex
Anterolateral	Anterolateral	

Pericardium: Normal, Thickened, Effusion: mild, moderate, severe, Tamponade **Pleura:** nl,effusion

Post Intervention Follow-up Study: See additional report, No Change, **Ventricular Fxn:** Global FXN: Improved, Decreased, Regional FXN: Improved, Decreased, **Valve FXN:** Native Valve: no change, improved, Normal Prosthetic Valve type_____ nl fxn?: Y/N, Valve repair: 0 leak, 1+,2+,3+, Valve area___

3.Comments:_____

4.Summary:_____

5.Complications: None,details:_____

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Valves	Annulus	Stenosis	Area/ Gradient	Regurg	Leaflet Morphology	Leaflet Motion
Aortic Valve	normal, dilated, calc, bioprosth, mechanical	No, mild, Mod, severe		0, 1+, 2+, 3+ 4+	Nl, Calc, Veg, Perf, Bicuspid Thickened,	Nl, Prolapse Flail, Restricted NCC/RCC/LCC
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Right atrium	normal, dilated	y/n	y/n	y/n	y/n
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Location: ICU/OR/ER/PACU, Phys requesting Echo _____, Examiner_____
Indication_____ CPT Codes_____ICD-9 codes_____
Intubated: Y/N, Sedated: Y/N, Insertion: Easy/Difficult /Failed Probe Type: pediatric/ biplane/multiplane/ epicardial/epiaortic Modalities: 2D/CFM/PWD/CWD/Contrast/Pharm/Stress Tape/OD #_____

2.Echocardiographic and Doppler Measurements

Aorta	Size	Diam (cm)	Dissection	Plaque thick (mm)	Plaque mobile
Ascending Ao	nl, dilated, aneur		y/n	0-3, >3	y/n
Ao Arch	nl, dilated, aneur		y/n	0-3, >3	y/n
Descending Ao	nl, dilated, aneur		y/n	0-3, >3	y/n

Valves	Annulus	Stenosis	Area/ Gradient	Regurg	Leaflet Morphology	Leaflet Motion
Aortic Valve	normal, dilated, calc, bioprosth, mechanical	No, mild, Mod, severe		0, 1+, 2+, 3+ 4+	Nl, Calc, Veg, Perf, Bicuspid Thickened,	Nl, Prolapse Flail, Restricted NCC/RCC/LCC
Mitral Valve	normal, dilated, calc, bioprosth, mechanical	No, mild, Mod, severe		0, 1+, 2+, 3+ 4+	Nl, Calc, Veg Perf, Myxom Thickened	Nl, Prolapse, Flail, SAM, Restricted A_1,A_2,A_3/ P_1,P_2,P_3
Tricuspid	normal, dilated, calc, bioprosth, mechanical	No, mild, Mod, severe		0, 1+, 2+, 3+ 4+	Nl, Calc, Veg Perf, Myxom Thickened	Nl, Prolapse Flail, Restricted

Atria	Size	SEC (smoke)	Thrombus	Tumor	Device
Right atrium	normal, dilated	y/n	y/n	y/n	y/n
Left atrium	normal, dilated	y/n	y/n	y/n	y/n

Left atrial appendage:

Interatrial Septum: Morphology: Normal, Aneurysm Lipomatous Hypertrophy, PFO, ASD (Primum, Secundum, Sinus Venosus, AV Canal) Shunt: (r->l, l->r, bidirectional)

Interventricular Septum Morphology: Normal, Hypertrophy, Shift, Defect (membr, musc), Shunt: (r->l, l->r)

Ventricles	Cavity size	Cavity Dimension	Hypertrophy	Thrombus	Global FXN	EF
RV	normal, dilated		y/n	y/n	Nl, ↓, ↓↓,↓↓↓	
LV	normal, dilated		y/n	y/n	Nl, ↓, ↓↓,↓↓↓	

Regional Function: (1=normal, 2= hypokinetic, 3= akinetic, 4=dyskinetic)

Basal Segments	Mid Segments	Apical Segments
Anterior	Anterior	Anterior
Anteroseptal	Anteroseptal	Septal
Inferoseptal	Inferoseptal	Inferior
Inferior	Inferior	ateral
Inferolateral	Inferolateral	True apex
Anterolateral	Anterolateral	

Pericardium: Normal, Thickened, Effusion: mild, moderate, severe, Tamponade **Pleura:** nl,effusion

Post Intervention Follow-up Study: See additional report, No Change, **Ventricular Fxn:** Global FXN: Improved, Decreased, Regional FXN: Improved, Decreased, **Valve FXN:** Native Valve: no change, improved, Normal Prosthetic Valve type_____ nl fxn?: Y/N, Valve repair: 0 leak, 1+,2+,3+, Valve area___

3.Comments:_____

4.Summary:_____

5.Complications: None,details:_____

Sample Perioperative Echo Report

1. Demographic and Other Identifying Information

Name_____ Unit #_____DOB_____ M/F Date_____ Time____
Location: ICU/OR/ER/PACU, Phys requesting Echo _____, Examiner_____
Indication_____ CPT Codes_____ICD-9 codes_____
Intubated: Y/N, Sedated: Y/N, Insertion: Easy/Difficult /Failed Probe Type: pediatric/ biplane/multiplane/ epicardial/epiaortic Modalities: 2D/CFM/PWD/CWD/Contrast/Pharm/Stress Tape/OD #_____

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Aorta	**Size**	**Diam (cm)**	**Dissection**	**Plaque thick (mm)**	**Plaque mobile**
Ascending Ao	nl, dilated, aneur		y/n	0-3, >3	y/n
Ao Arch	nl, dilated, aneur		y/n	0-3, >3	y/n
Descending Ao	nl, dilated, aneur		y/n	0-3, >3	y/n

Valves	**Annulus**	**Stenosis**	**Area/ Gradient**	**Regurg**	**Leaflet Morphology**	**Leaflet Motion**
Aortic Valve	normal, dilated, calc, bioprosth, mechanical	No, mild, Mod, severe		0, 1+, 2+, 3+ 4+	Nl, Calc, Veg, Perf, Bicuspid Thickened,	Nl, Prolapse Flail, Restricted NCC/RCC/LCC
Mitral Valve	normal, dilated, calc, bioprosth, mechanical	No, mild, Mod, severe		0, 1+, 2+, 3+ 4+	Nl, Calc, Veg Perf, Myxom Thickened	Nl, Prolapse, Flail, SAM, Restricted A_1,A_2,A_3/ P_1,P_2,P_3
Tricuspid	normal, dilated, calc, bioprosth, mechanical	No, mild, Mod, severe		0, 1+, 2+, 3+ 4+	Nl, Calc, Veg Perf, Myxom Thickened	Nl, Prolapse Flail, Restricted

Atria	**Size**	**SEC (smoke)**	**Thrombus**	**Tumor**	**Device**
Right atrium	normal, dilated	y/n	y/n	y/n	y/n
Left atrium	normal, dilated	y/n	y/n	y/n	y/n

Left atrial appendage:

Interatrial Septum: Morphology: Normal, Aneurysm Lipomatous Hypertrophy, PFO, ASD (Primum, Secundum, Sinus Venosus, AV Canal) Shunt: (r->l, l->r, bidirectional)

Interventricular Septum Morphology: Normal, Hypertrophy, Shift, Defect (membr, musc), Shunt: (r->l, l->r)

Ventricles	**Cavity size**	**Cavity Dimension**	**Hypertrophy**	**Thrombus**	**Global FXN**	**EF**
RV	normal, dilated		y/n	y/n	Nl, ↓, ↓↓,↓↓↓	
LV	normal, dilated		y/n	y/n	Nl, ↓, ↓↓,↓↓↓	

Regional Function: (1=normal, 2= hypokinetic, 3= akinetic, 4=dyskinetic)

Basal Segments	**Mid Segments**	**Apical Segments**
Anterior	Anterior	Anterior
Anteroseptal	Anteroseptal	Septal
Inferoseptal	Inferoseptal	Inferior
Inferior	Inferior	ateral
Inferolateral	Inferolateral	True apex
Anterolateral	Anterolateral	

Pericardium: Normal, Thickened, Effusion: mild, moderate, severe, Tamponade **Pleura:** nl,effusion

Post Intervention Follow-up Study: See additional report, No Change, **Ventricular Fxn:** Global FXN: Improved, Decreased, Regional FXN: Improved, Decreased, **Valve FXN:** Native Valve: no change, improved, Normal Prosthetic Valve type_____ nl fxn?: Y/N, Valve repair: 0 leak, 1+,2+,3+, Valve area___

3.Comments:_____

4.Summary:_____

5.Complications: None,details:_____

OBJECTIVES

Thursday, February 14, 2008

Advanced Echocardiography I

Ventricular Function
Moderator: Shernan SK (Troianos)

Advanced Challenging Cases: K Grichnik, C Troianos
At the conclusion of this lecture, the participant will:
1) Understand the general scope of advanced cases they should be able to understand and diagnose. Cases will be presented along with representative videos and sample questions. On Saturday morning, these cases will be reviewed in detail.

Advanced Systolic Function – AT Cheung
At the conclusion of this lecture, the participant will:
1) Be prepared to apply echocardiography for assessment of LV systolic function
2) Be able to recognize the advantages and disadvantages of various techniques

Advanced Diastolic Function: "Beyond the E- and A-Wave" – M Swaminathan
At the conclusion of this lecture, the participant will:
1) Be able to differentiate systolic function from diastolic function as a basis for pathophysiology
2) Be able to assess diastolic dysfunction with various echocardiographic techniques
3) Recognize the advantages and limitations of each technique

Tissue Doppler/Stress/Strain – NJ Skubas
At the conclusion of this lecture, the participant will:
1) Understand techniques for acquiring Doppler tissue imaging, strain, and strain rate
2) Be able to describe applications for Doppler tissue imaging, strain, and strain rate in the evaluation of myocardial performance

3D and 4D Echocardiography – D Shook
At the conclusion of this lecture, the participant will be able to:
1) Describe the techniques for acquiring and reconstructing 3D echocardiographic images
2) Describe the techniques for acquiring Real Time 3D echocardiographic images
3) Identify perioperative applications of 3D echocardiography

Case Discussions with Faculty – Cheung, Swaminathan, Skubas, Shook, Shernan
Examples and discussion advanced systolic, diastolic function, stress/strain and 3D echo will be presented

Echocardiography for Heart Failure and Cardiomyopathies

Moderator: AD Maslow

Echocardiographic Evaluation of Cardiomyopathies – AD Maslow
At the conclusion of this lecture, the participant will be able to:
1) Identify subcategories of cardiomyopathies
2) Apply echo for quantitative analysis of LV function in patients with cardiomyopathies

OBJECTIVES

Pericardial Disease – EG Avery
At the conclusion of this lecture, the participant will be able to:
1) Identify subcategories of pericardial disease
2) Apply Echo for quantitative analysis of LV function in patients with pericardial disease

Intraoperative Echo for Ventricular Assist Devices and Heart Transplantation - TM Burch
At the conclusion of this lecture, the participant will understand:
1) The risk of VAD
2) Critical information required to manage the risk of VAD
3) How Echo can help manage the risk of VAD

Advanced Echocardiography II: Aortic Valve – Advanced
Moderator: BA Bollen

Timing of Surgery/Intervention in Asymptomatic Aortic Valve Disease - AE Weyman
At the conclusion of this lecture, the participant will be able to:
1) Describe the important factors in evaluating aortic valve disease and the timing of surgical intervention
2) Identify when intervention is necessary in patients with asymptomatic aortic valve disease

Patient Prosthetic Mismatch and Pressure Recovery after Aortic Valve Replacement– I Kronzon
At the conclusion of this case discussion, the participants should:
1) Have a better understanding of the phenomenon and significance of pressure recovery
2) Have a better understanding of patient prosthetic mismatch
3) Have a better understanding of the different perspectives in the perioperative treatment of and decision making in patients with small aortic root dimensions

Aortic Valve Replacement or Repair – NG Smedira
At the conclusion of this lecture, the participant will be able to:
1) Explain how echocardiography can be used to evaluate normal aortic valve anatomy
2) Explain how echocardiography can be used to evaluate aortic valve pathology and function
3) Describe the impact of echocardiography on surgical decision making during aortic valve procedures
4) Identify the surgical options available in patients with aortic valve disease
5) Understand the technical issues, which must be addressed to insure success of each procedure
6) Compare the advantages and disadvantages of each of these procedures
7) Describe the potential complications of each of these surgical procedures

Case Discussions with Faculty - Bollen, Weyman, Kronzon, Smedira
At the conclusion of this case discussion, the participants should:
1) Have a better understanding of the roles that cardiologists, surgeons and anesthesiologists play in the diagnosis and surgical treatment of aortic valve disease
2) Have been introduced to the specific technical and intellectual expertise that each discipline brings to the care of patients with aortic valve disease
3) Have a better understanding of the different perspectives in the perioperative treatment of patients with advanced aortic valve disease

Advanced Hemodynamic Workshop
Moderator: Cheung, Maslow

Review of Advanced Hemodynamic Principles and Quantitative Doppler – A Maslow
Upon completion of this lecture, the participant should understand:
1) The physics and formulae used in Quantitative Doppler Echocardiography
2) How Quantitative Doppler Echocardiography and knowledge of Hemodynamic Principles can be used
3) How to calculate intracardiac velocity, flow, chamber pressures and valve areas
4) The limitations of Quantitative Doppler Echocardiography

Advanced Hemodynamic Case Discussions – Cheung, Shernan; Konstadt, Koch, Maslow
Upon completion of this lecture, the participant should understand:
1) How to obtain estimates of intracardiac velocity, flow, chamber pressures and valve areas using a Case Discussion format
2) The clinical applications of measurements and calculations obtained by Quantitative Doppler Echocardiographic techniques

Friday, February 15, 2008

Advanced Mitral Valve
Moderator: BA Bollen

Your Patient is Scheduled for Mitral Valve Repair but Presents with Only 2+ Regurgitation by Intraoperative TEE: What Now?- S. Konstadt
At the end of this lecture the participant will understand:
1) Physiology of mitral regurgitation and anesthesia
2) Pharmacologic maneuvers to manipulate mitral regurgitation
3) Decision-making in mitral valve surgery

Ischemic Mitral Regurgitation - S Aronson
At the conclusion of this lecture, the participant will understand:
1) Anatomy and function of the mitral valve apparatus
2) The definition of ischemic mitral regurgitation
3) Changes of the MV apparatus associated with chronic myocardial ischemia
4) Long-term strategies in the surgical management of ischemic MR

Surgical Repair of the Mitral Valve - D Adams
At the conclusion of this lecture, the participant will understand:
1) Surgical anatomy of the mitral valve apparatus
2) The surgeon's systematic examination of the mitral valve apparatus
3) Correlation of surgical inspection with the intraoperative echo examination
4) Surgical considerations and options in MV repair
5) Causes of unsuccessful MV repair

OBJECTIVES

Lecture: Case Discussions with Faculty - Bollen, Konstadt, Aronson, Adams

At the conclusion of this case discussion, the participants should:

1) Have a better understanding of the roles that cardiologists, surgeons and anesthesiologists play in the diagnosis and surgical treatment of mitral valve disease
2) Have been introduced to the specific technical and intellectual expertise that each discipline brings to the care of patients with mitral valve disease
3) Have a better understanding of the different perspectives in the perioperative treatment of patients with advanced mitral valve disease

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Role of Echocardiography in Perioperative Clinical Decision-making: Part I

Moderator: RM Savage

Role of Echocardiography in Percutaneous Approaches to Valve Dysfunction - RM Savage

At the conclusion of this lecture, the participant will understand:

1) Current practices related to valve procedures in the catheterization lab
2) Echocardiographic goals in this patient population
3) Indications and contraindications for percutaneous procedures

Mitral Valve Dysfunction after Mitral Valve Repair - S Shernan

At the conclusion of this lecture, the participant will understand:

1) Mechanisms of post-repair MS and MR
2) Efficient MS/MR severity assessment methods in post-CPB MV repair
3) Surgical considerations in re-repair of the mitral valve
4) Long term outcomes of different MV repair techniques

Luncheon Series

Future of Cardiac Surgery – D Adams

At the conclusion of this lecture, the participant will understand:

1) Demographic trends impacting the future of cardiovascular surgery
2) Evolving technologies that will impact the management of cardiovascular disease
3) Complications of newer technologies and their perioperative management
4) Future of training in the cardiovascular disciplines

Role of Echocardiography in Perioperative Clinical Decision-making: Part II

Moderator: RM Savage

How to Use Echocardiography to Help Wean from Bypass - T. Burch

At the conclusion of this lecture, the participant will understand:

1) Anesthetic-hemodynamic considerations in successful weaning from CPB
2) Etiologies of myocardial dysfunction during separation from CPB
3) The use of intraoperative echo in guiding the separation from CPB
4) The therapeutic options available in difficult weaning from CPB and their indications

OBJECTIVES

Unanticipated Ascending Aortic Dilatation: How Big is Too Big? - AT Cheung
At the conclusion of this lecture, the participant will understand:
1) The integrated anatomy of the aortic root complex
2) Etiologies of ascending root dilatation
3) Surgical considerations in unanticipated ascending aortic dilatation
4) Intraoperative echo in directing the management of ascending aortic dilatation

Echocardiographic Assessment of Myocardial Viability - M Swaminathan
At the conclusion of this lecture, the participant will:
1) Be able to illustrate the need to identify viable myocardium for acute care
2) Be able to describe the methodologies available to assess viability
3) Be able to evaluate the functionability of echocardiography for evaluating viability perioperatively

Echocardiography for Adult Congenital Heart Disease – I Russell
At the conclusion of this lecture, the participant will understand:
1) Understand the common adult congenital pathologies presenting for primary or repeat cardiac surgery
2) Understand management of congenital heart disease patients for non-cardiac surgery when they are partially corrected, palliated, or awaiting correction
3) Understand echocardiographic anatomy of common congenital disease processes

Stump the Panel: Unusual Cases from the Faculty - Savage, Burch, Cheung, Swaminathan, Russell
At the conclusion of this panel, the participant will:
1) Gain knowledge of unusual echocardiographic diagnoses
2) Develop a process for evaluating echocardiograms when the diagnosis is difficult

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"Test Yourself"
Moderator: Zvara

At the conclusion of this workshop, the participant will:
1) Have been assessed on a broad range of echocardiographic knowledge in a simulated testing environment
2) Be able to determine their areas of relative strength and weakness in preparation for the standardized echocardiography competency examinations
3) Be able to place their relative testing knowledge against other participants taking the mock examination
4) Devise test study strategies, which may help guide preparation for standardized echocardiography examinations

Saturday, February 16, 2008

You're the Intraoperative Echocardiography Consultant
Moderators: Shernan and Hartman

You're the Intraoperative Echocardiography Consultant:
Upon completion of this session, the participant will have a better understanding of:
1) How to be a perioperative echocardiography consultant
2) The utility of echocardiography for perioperative decision-making through the use of interactive case discussions
3) Unusual cases and the application of perioperative ultrasound to clinical care of patients with cardiovascular disease

NOTES

PRODUCT DESCRIPTIONS

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The American Society of Echocardiography (ASE)

1500 Sunday Drive, Suite 102 Raleigh, NC 27607
Phone: 919-861-5574
Fax: 919-787-4916
Website: www.asecho.org

The American Society of Echocardiography (ASE) is an organization of professionals committed to excellence in cardiovascular ultrasound and its application to patient care through education, advocacy, research, innovation and
service to our members and the public. Information on ASE membership, echocardiography standards, research grants, patient brochures, and Association educational programs are available. Join ASE in Toronto June 7-11, 2008 for the 19th Annual Scientific Sessions!

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Lippincott Williams & Wilkins
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Gary.F.White@wolterskluwer.com

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PRODUCT DESCRIPTIONS

National Board of Echocardiography, Inc.

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NOTES

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FUTURE MEETINGS

2008

March 9-14 — **13th Annual Update on Cardiopulmonary Bypass**
Fairmont Chateau Whistler
Whistler, BC, Canada

June 18-22 — **30th Annual Meeting & Workshops**
Vancouver Convention & Exhibition Centre
Vancouver, BC, Canada

2009

February 9-14 — **12th Comprehensive Review of Perioperative Echo**
Sheraton San Diego Hotel and Marina
San Diego, CA

March 15-20 — **14th Annual Update on Cardiopulmonary Bypass**
Fairmont Chateau Whistler
Whistler, BC Canada

April 18-22 — **31st Annual Meeting & Workshops**
Grand Hyatt San Antonio
San Antonio, Texas

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