SCA NEWS

SOCIETY OF CARDIOVASCULAR ANESTHESIOLOGISTS
Knowledge • Care • Investigation

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SOCIETY OF CARDIOVASCULAR ANETHESIOLOGISTS

PRESIDENT'S MESSAGE

Amanda Fox MD, MPH

President, Society of Cardiovascular Anesthesiologists

Dear Members of the Society of Cardiovascular Anesthesiologists (SCA),

On Monday, April 28, Dr. Katherine Glas passed the gavel to me as the incoming SCA President. I want to recognize and thank Dr. Glas for her excellent leadership of the SCA as well as for her many other contributions to the society over the years. I look forward to continuing to work with her over the next two years as she serves with me on the SCA's Executive Committee as the Immediate Past President. Also, I want to acknowledge the other two members of the SCA's Executive Committee, Dr. Douglas Shook (President Elect) and Dr. Mary Beth Brady (Secretary/Treasurer) for their new roles on the Executive Committee. We look forward to working with the SCA's Board of Directors, our Committee Members, the administrative management team (Veritas management company), and you—the SCA's members—leveraging all our strengths to further advance the society.

It was fantastic to see so many of you at the SCA's 47th Annual Meeting and Workshops in Montreal, Canada! Over 1400 people attended! I would also like to celebrate this year's Thoracic Anesthesiology Symposium and to acknowledge the TAS Program Committee members, the symposium's Chair Dr. Rebecca Klinger, and the Vice Chair Dr. Emily Teeter for their excellent work.

My heartfelt thanks to the Scientific Program Committee members for organizing such a cutting-edge and engaging educational program for the Annual Meeting and Workshops in Montreal. Thanks to the Annual Meeting's Program Chair Dr. Jonathan Ho, Vice Chair Dr. Stephanie Ibekwe, and Workshop and PBLD Chair Dr. Tara Brakke for their unwavering commitment to putting all of this together.

Finally, I want to give a big shout-out to Dr. Jessica Brodt for her leadership in planning a fantastic SCA Gala that was held on April 26th at the majestic St. James Theater in Montreal. Thank you also to Dr. Isobel Russel, Chair of the SCA's Endowment Council, for leading in the making of a beautiful tribute to the late Dr. Michael Cahalan. (https://scahq.org/endowment/planned-giving/michael-k-

PRESIDENT'S MESSAGE *continued*

cahalan-legacy-circle/). The opportunity to celebrate Dr. Cahalan's life, his numerous contributions to the field of cardiac anesthesiology, and his generous mentorship of many other leaders in our subspecialty made this a particularly special SCA Gala. I look forward to seeing you all at SCA's 48th Annual Meeting and Workshops April 23-26, 2026 in Nashville, TN.

In addition to the Annual Meeting and Workshops and the Thoracic Anesthesiology Symposium, the SCA offers a range of other excellent educational meetings and offerings, including the SCA ECHO meeting, the SCA ECHO Board Review, the Anesthesia Review Cards (ARC), SCA University, and additional projects created and offered through the Online Education Committee. All educational content that is online at SCA University, including CME offerings, are free for all of our members.

Like many of you, I deeply enjoy networking with one another and learning about the latest innovations in cardiac anesthesia through these opportunities. The SCA shines brightly amongst professional societies in large part because of the dedication and investment of SCA's members in advancing our subspecialty. I am honored to serve as your new President. I take on this role with our society being in sound financial standing. The Endowment Council and its Chair Dr. Isobel Russell are hard at work fundraising for the SCA's expanded future aims in advancing our priorities in research, exploring the potentials of AI, and broadening international collaborations. I'm also looking forward to working together on increasing our educational endeavors and offerings, and on advancing community-building and professional engagement opportunities within our subspecialty. Please feel free to contact me, or members of the Executive Committee or Board of Directors, about any ideas or concerns as we continue to grow and move further forward as a society.

With gratitude,

Amanda Fox, MD
SCA President, 2025-2027

SCA ANNUAL MEETING *Highlights*

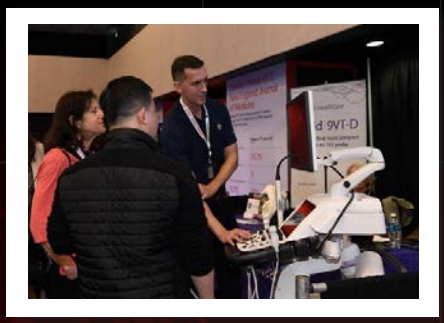
Jonathan Ho, MD
Annual Meeting Chair

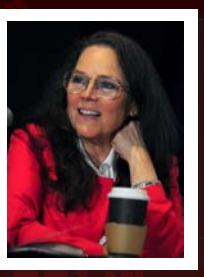
Stephanie Ibekwe, MD
Annual Meeting Vice-Chair

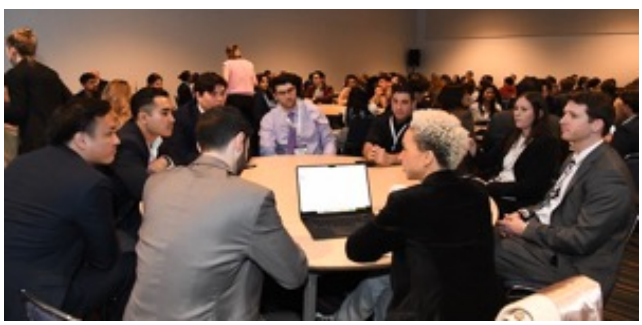
Rebecca Klinger, MD
Thoracic Symposium Chair

A huge thank you to all who attended the 2025 SCA Annual Meeting and Thoracic Anesthesia Symposium in Montreal, Canada and made it a huge success. We hope you enjoyed the meeting as much as we did!

Meeting Memories







Special Thanks

KEYNOTE SPEAKER

Christopher A. Troianos, MD, FASE, FASA

The Importance of Physician Leadership in Today's Healthcare Challenges

J. EARL WYNANDS LECTURER

Andre Denault, MD, PhD

Hemodynamically Unstable, Why?

Thank You

FOR JOINING US AT THE GALA EVENT!

We extend our heartfelt thanks to everyone who attended the Gala Event at the stunning St. James Theatre in Montreal during this year's Annual Meeting. Your presence and support helped make the evening a tremendous success.

It was truly a night to remember—filled with inspiring speakers, captivating performances, and a lively silent auction. Most importantly, together we raised valuable funds for the SCA Endowment.

We're grateful for your continued support and can't wait to welcome you to the 2026 Annual Meeting in Nashville, Tennessee!

SCA GALA 2025

SOCIETY OF CARDIOVASCULAR ANESTHESIOLOGISTS
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Congratulations to Our Esteemed Colleagues!

ANNUAL SOCIETY AWARDS AND RECOGNITION

Presidential Outstanding Service Award
Glenn P. Gravlee, MD (right)

John Hinckley Outstanding Service Award
Charles P. Boinske, CFA

Distinguished Service Award
Michael Cahalan, MD
(Accepted by Marianne Cahalan)

Presidential Citation
Jessica Brodt, MD (left)

Presidential Citation
Jennifer Hargrave, DO (left)

Early Career Investigator Awardees with Program Leadership

Jr. Resident Scholarship Recipients with Program Leadership

Kaplan Leadership Development Award
Gina Linganna, MD (left)

Kaplan Leadership Development Award
Nadia Hensley, MD

DISTINGUISHED SERVICE AWARD

The Distinguished Service Award is given to an individual who has made significant contributions to the specialty of cardiovascular anesthesiology through research, education, service, or any combination of these activities.

Michael K. Cahalan, MD

PRESIDENTIAL OUTSTANDING SERVICE AWARD

The Presidential Lifetime Outstanding Service Award is given to an anesthesiologist who has made outstanding long-term contributions to the Society.

Glenn P. Gravlee, MD

JOHN HINCKLEY OUTSTANDING SERVICE AWARD

The John Hinckley Outstanding Service Award will be given to a non-anesthesiologist who has made an outstanding contribution to the Society.

Charles P. Boinske, CFA

PRESIDENTIAL CITATIONS

The Presidential Citation Award is given at the discretion of the President (and/or Executive Committee) to an individual who has made an important contribution to the field of cardiovascular anesthesia through 1) an early career accomplishment; 2) research contribution; 3) education contribution; 4) DEI contribution through advancement of minority interests; and/or 5) service to the SCA.

Jessica Brodt, MD
Jennifer Hargrave, DO

SCA Research Grant & Scholarship Recipients

The SCA is excited to announce the following 2025 grant and scholarship winners.

Jr. Resident Scholarship Recipients

Biodun Adeniyi, MD
Susana Arango Arango, MD
Jane-Frances Aruma, MD
Richard Beckett-Ansa, MD
Cheng-Hao Jacky Chen, MD
Robert Hawthorne, MD, PhD
JooEun Kang, MD
Laura Mendez Pino, MD
Kwame Wiredu, MD

Kaplan Leadership Development Award

Nadia Hensley, MD – *Executive Leadership in Healthcare Certificate of Specialization at Harvard T. H. Chan School of Public Health*

Regina Linganna, MD – *Master's in Medical Education*

Early Career Investigator Award

Serena S. Dasani, MD, MBA - *Advocating for Healthcare Policy Reform: Addressing Reimbursement Gaps for Rescue TEEs Performed by Cardiovascular Anesthesiologists*

Nicolas T. Quach, MD - *Go with the (optical) Flow: A Novel Method for the Echocardiographic Quantification of Right Ventricular Function Using an Explainable Deep-learning Assisted Approach*

Anjan Saha, DO - *Pre-Transplant Blood Transfusion and Outcomes in Lung Transplant Recipients*

Benjamin Steinhorn, MD, PhD - *A Novel Method for Real-time Measurement and Clinical Application of Mean Systemic Filling Pressure in the Cardiac ICU*

Kwame Wiredu, MD, PhD - *Systemic Inflammation and Metabolic Dysregulation after Cardiac Surgery: a pathway to Delirium in the Cognitively Vulnerable Patient*

Sherman Yu, MD - *Right Ventricular Pressure-Strain Loop-Derived Global Myocardial Work in Patients Undergoing Left Ventricular Assist Device Implantation*

In-Training Grant / *$15,000 for 1 year*

Kitae Chang, MD, MS – *Adjustable Bioadhesive Ultrasound (ABAUS) for Continuous Monitoring of Impella LVAD Patients in the ICU*

Joyce Wahr Starter Grant / *$25,000 per year for 2 years*

Anastasia Katsiampoura, MD, PhD – *Elucidating Anti-inflammatory Roles of CD39 and T regulatory cells with Cardiopulmonary Bypass*

Megan Henley Hicks, MD – *Continuous Glucose Monitoring in Patients Undergoing Cardiopulmonary Bypass*

Mid-Career Research Grant / *$50,000 per year for 2 years*

Zoel Augusto Quiñónez, MD – *Predicting Prolonged Hospitalization after Congenital Heart Surgery Using Medicaid Administrative Claims and Machine Learning*

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RECOGNITION OF SERVICE

SCA Executive Committee, 2025-2027

Amanda A. Fox, MD
President

Mary Beth Brady, MD
Secretary/Treasurer

Douglas C. Shook, MD
President-Elect

Kathryn E. Glas, MD, MBA
Immediate Past-President

Outgoing Board Members

Kathryn E. Glas, MD, MBA, FASE
SCA President, 2023-2025

Andrew D. Shaw, MB, FRCA, FFICM, FRCPC
SCA Immediate Past President, 2023-2025

Douglas C. Shook MD, FASE
Secretary/Treasurer, 2023-2025

Amanda A. Fox, MD, MPH
President-Elect, 2023-2025

Hilary Grocott, MD
2023-205
Director-at-Large

Daryl Oakes, MD
2023-2025
Director-at-Large

Annemarie Thompson, MD
2022-2025
Director-at-Large

Lisa Rong, MD
2023-2025
Director-at-Large

Incoming Board Members

Mary Beth Brady, MD, FASE
Secretary/Treasurer, 2025-2027

Daryl Oakes, MD
2025-2027
Director-at-Large

Charles B. Nyman, MBBCh
2025-2028
Director-at-Large

Jacob Raphael, MD
2025-2027
Director-at-Large

The award is designed to assist cardiothoracic and vascular anesthesiologists in their career by granting funding to further their leadership development through coursework and leadership-specific studies. The Kaplan Leadership Award will be adjusted accordingly to offer an aggregate of $5,000 to either one recipient or divided among two.

$5,000/$2,500 from the SCA Endowment, with a $5,000/$2,500 match from the applicant's institution to fund a leadership education strategy.

Click here for more information on this award. Applications will be accepted this fall. Questions? Please contact us at operations@scahq.org.

KAPLAN RECOGNITION

During the 2025 SCA Annual Meeting, the Endowment Council recognized past Kaplan awardees.

SCA 2025 Annual Meeting OnDemand at Your Convenience — Anytime, Anywhere.

Get trusted Society of Cardiovascular Anesthesiologists meeting content in an easy-to-navigate platform designed to save you time. SCA 2025 Annual Meeting OnDemand is packed with hours of value, access to top presentations from the event, and the opportunity to earn 59 CME. Gain access to clinical practice tips, advancements in Cardiovascular anesthesiology, case-based reviews, and over 36 Sessions covering topics including:

- Recent advancements in cardiac surgery and anesthesia
- Patient-centered care and clinical outcomes
- Tools to strengthen multidisciplinary collaboration

To purchase, click HERE!

Check it out!

Journal of Anesthesia & Analgesia — How to View Free Access Articles

Below are links to the three SCA sections of the A&A Journal. Each month, these links automatically update with new publications. "Free Access" articles will have a "Free" tag just below the article details. After one year, all A&A articles become complimentary.

Cardiovascular and Thoracic Anesthesiology

Cardiovascular Pathophysiology and Outcomes

Hemostasis and Thrombosis

Call for Volunteers Coming this Fall! April 2026 – April 2028 Term Selection

Support your Society's strategic goals and initiatives by serving one of its 40-plus committees and sub-committees! The Call for Volunteers will be open this October to fulfill the 2026-2028 term. Watch your in-box later this summer for details.

For questions related to the Call for Volunteers, please email committees@scahq.org

The Call for Nominations for the SCA Board of Directors Opening Soon!

Show your commitment to the value of the Society of Cardiovascular Anesthesiologists to shape its future! You may nominate yourself or a committed SCA colleague.

More details are forthcoming in the next month.

2026 Annual Society Awards Call for Recommendations Opening August 1st

SCA Annual Awards

The SCA encourages its members to honor those who have made a significant impact within the Society and the sub-specialty of cardiovascular anesthesiology by recommending them for one of its annual awards. The call will be open August 1 – September 15, 2025. Watch your in-box and the SCA Website for details.

The Distinguished Service Award

- Honors an individual who has made a meaningful contribution to the ***field of cardiovascular anesthesiology*** through research, education, or service that has produced a significant impact in the field.
- This individual does not have to be an anesthesiologist but must be a member of the SCA.

The Presidential Outstanding Service Award

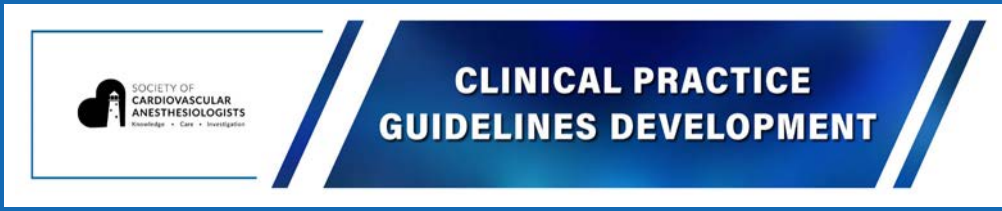
- Honors an individual who has made outstanding, long-term contributions to the ***Society of Cardiovascular Anesthesiologists (SCA).***
- This individual must be an anesthesiologist and a member of the SCA.

The John Hinckley Outstanding Service Award

- Honors an individual who has contributed to or advanced the ***field of cardiovascular anesthesiology*** through education, research, or innovative clinical work.
- This individual must be a ***non-physician.*** Membership in the SCA is not required.
  - Examples of possible recipients include perfusionists, blood bank personnel, etc.

A listing of past Society awardees may be found on the SCA website: **CLICK HERE.**

Funding Opportunity from the Guidelines and Standards Committee!



Clinical Practice Guidelines Development Submissions NOW Open.

The Call for Clinical Practice Guidelines (CPGD) Development funding is now open. Up to four grants per year will be awarded. Each selected applicant will be awarded up to $15,000.

Application Requirements:

1. Introduction/Background
2. Outline Objectives/Scope
3. Methodology: Describe the methodology for the project development
4. Budget and Justification
5. Contributors: Describe the roles of the key personnel and their qualifications primarily as it relates to the proposed work
6. Letter of support from SCA Sub-Committee and Parent Committee Chair through which the application is submitted
7. Conflict of Interest

Please Note: The application must be submitted through an SCA committee. The applicant must be an SCA member at the time of the application. We encourage SCA Committee and Subcommittee members to apply.

The committee will not consider applications from authors currently receiving funding for similar purpose, independent of source; however, they may serve as a co-author on a CPG development application. Applications for scoping reviews will be accepted.

Barriers to Women Advancing to Senior Leadership in Anesthesiology

Adam J. Milam, MD, PhD[1]; Belle Benanzea-Fontem, MD; Stephanie Ibekwe, MD, MBA, MPH, MS[3]
[1]Department of Anesthesiology and Perioperative Medicine, Mayo Clinic
[2]Department of Anesthesiology & Perioperative Medicine; University of California, Los Angeles
[3]Department of Anesthesiology, Baylor College of Medicine

Adam Milam, MD, PhD

Belle Benanzea-Fontem, MD

Stephanie Ibekwe, MD, MBA, MPH, MS

According to the Association of American Medical Colleges, only 25% of full professors are women, and 18% of department chairs and deans are held by women.[1] Furthermore, McMullen and colleagues found there are no women as editor-in-chief for anesthesiology journals, and women only represent 18% of all editorial board positions.[2] This lack of representation in senior roles exists despite women representing 51% of medical school applicants and 48% of medical school graduates. Mondal et al shed light on the systemic barriers that prevent women from advancing to senior leadership positions within anesthesiology (i.e., mid-career stall) and presented solutions to address these barriers.[3]

The lack of advancement of women to senior leadership is problematic based on our moral and ethical obligations, Mondal et al explains, and may negatively impact patient care and education.[3] Women physicians on average, provide more complex care to patients, spend more time on patient care, and spend more time focused on education.[3] Essentially women are leading two of the three shields in academic medicine: education and clinical practice, yet women are less likely to be in senior leadership roles, are compensated less, and have fewer opportunities for first authored publications. Mondal and colleagues discussed the harm of mid-career stall: higher rates of burnout, lower satisfaction with work-life integration, and a higher likelihood of leaving the field of medicine.[3] They also highlighted intersectionality in the context of the consequences of mid-career stall: being minoritized as a woman, increasing attrition, and being denied promotions.

The systemic barriers faced by mid-career women physicians include the following:

Feedback	Feedback pertaining to behavior and personality for women versus competence and performance for men.
Mentorship	Women report facing more challenges identifying mentors and sponsors when compared to men.
Fitting In	Metrics, communication styles, and work attitudes were designed based on norms and values of men.
Recognition	Women have a disproportionate burden of non-promotable work, like community-building efforts, and lower tier education roles versus program directors and editorial roles for men.
Bias/Mistreatment	Women are more likely to experience mistreatment compared to their male colleagues and there are gender inequities in compensation leading higher rates of burnout.
Time Demands	Women physicians delay pregnancy leading to childbirth during the midcareer stage resulting in increasing concurrent demands.

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Burnout, attrition, and stalling career advancement are the sequelae of these systemic barriers. Given the invaluable role women play in medicine and medical education Mondal et al. provides recommendations to address these barriers:[3]

1. Provide more opportunities for mentorship and sponsorship
2. Invest in training for women seeking opportunities for professional skill building
3. Identify a niche and prioritize high-yield professional development activities, and
4. Diversify promotion criteria at Academic Institutions with less emphasis on publication-based metrics.

While these recommendations are reasonable, recommendations two and three place the burden on overburdened women colleagues. These recommendations also do not address the systemic failure that pressures women to delay familial desires to ensure time for mid-career progression. Nevertheless, with these initial actions put into place, our specialty may make some preliminary advancement in alleviating these barriers.

References

1. Association of American Medical Colleges. Active physicians by sex and specialty, 2021. Accessed August 3, 2023. https://www.aamc.org/data-reports/workforce/data/active-physicians-sex-specialty-2021.
2. McMullen, K., Kraus, M. B., Kosiorek, H., & Harbell, M. W. (2022). Representation of women as editors in anesthesiology journals. *Anesthesia & Analgesia*, 134(5), 956-963.
3. Mondal, S., Oakes, D., Humphrey, T., Kolarczyk, L., & Trzcinka, A. (2022). Women in anesthesiology and the mid-career stall: why they are not advancing into senior leadership. *Anesthesia & Analgesia*, 10-1213.

SPOTLIGHT

Awesome Woman Interview

Jennie Ngai, MD

NYU Langone Health
New York, NY

Looking back at my life, I attribute much of it to being in the right place at the right time. Having a spontaneous conversation with my teacher led me towards science and medicine as a high school student, while all of my friends were doing arts and music. A conversation with a non-anesthesiology attending faculty member as a medical student on a different rotation led me to anesthesiology. Anesthesiology was not a mandatory rotation and I would have likely passed right by it. A conversation with an anesthesiologist offering a cardiac anesthesiology fellowship position when I was instead considering job offers.

Many people would argue that luck does not make a person's life, but rather self-drive and diligence are more impactful. Perhaps it is both. It is whimsical to think that I float in the wind and see which direction the wind blows in. I say that about myself because I do not know what I want. Who knows what opportunities may arise? I want to see what life could look like at each fork in the road and decide at that moment which path to take.

1. What led you to become a Cardiovascular/Thoracic Anesthesiologist?
It seems very cliché, but during my medical school elective in anesthesiology, I was able to observe a cardiac surgery case. I do not remember very many details except that it was fascinating. During my surgical intern year, I had second thoughts about my career choice to become an anesthesiologist, and I considered doing surgery instead. I ultimately decided to not go into surgery, and I chose to continue my journey into anesthesiology.

Once I was in the cardiac OR as an anesthesiology resident, I loved how interactive it was and how so many things were going on at once. There seemed to be so much chaos. I loved the energy. Now a days I just want quiet, I tune everything out and focus on the problem at hand, especially during emergencies.

2. How did you hear about the SCA?
As a cardiothoracic fellow, my attendings encouraged me to submit a project to present. To my surprise, it was accepted and I went to the meeting to present it at a poster session. While I was there, I met many other cardiac anesthesiologists and attended many of the lectures. I learned so much!

3. What roles have you held for the society?
I have been a fellowship program director for many years, and as a result, I have been a member of the fellowship PD council. We have done a lot of great work on that council, advocating for the fellows, including improving the application process and working together with the critical care PDs. A diversity task force was created from the PD council, and as a member of the task force we were able to do research and publish papers on how to improve diversity in cardiac anesthesiology. Many fellowship applicants continue to ask what efforts and what recommendations do we have for cardiac anesthesiologists to become more inclusive. I have served as a mentor to residents and fellows through the SCA. I have also been a member of the newsletter committee, the clinical practice improvement committee, the division chief council, the newly formed ARC council, and the atrial fibrillation sub-committee, which i am the current chair.

4. What is one of your greatest achievements as a Cardiovascular/Thoracic Anesthesiologist?
As physicians, our greatest achievements should always involve patient care. We have some very scary moments, but there are times when we know that we have contributed

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to something great. I never feel that these clinical achievements are just our own. We work together as a team, with our surgical colleagues, with our nurses and perfusionists. We started the heart and lung transplant program at NYU in 2018. Majority of the cardiac anesthesiologists in our group had never done transplants, including myself. I was asked to be the director of the heart and lung transplant anesthesiology. I subsequently met with both surgeons for the heart and lung transplant programs, and individually they assured me that the program would start small and it may eventually grow in the future. Less than 5 years later, we were doing 150 transplants each year, even with the pandemic occurring in the middle of that! We were very successful with the initiation and growth of both programs and had great outcomes. One of the accolades came from a heart transplant surgeon. He was very impressed by our group of cardiac anesthesiologists' seemingly effortless ability to take care of transplant patients.

5. Do you have any advice for fellows and residents?
It is hard to predict how your career will turn out, or where you will end up. I always tell people to take every opportunity that comes their way. You never know what doors may open as a result of doing one thing. You may meet people along the way that may offer you opportunities that will lead to other opportunities!

Also, if there is something that you want to do – go after it. Ask the person in charge of the project if you can help out. If they say yes, then work hard on it, and it can lead to other opportunities. If they say no, they don't need help. Find another project and ask if you can help!

6. Have you experienced any difficulties as a woman in the field?
I have had to deal with many difficulties! The one that immediately comes to mind is regarding promotion. I had a conversation about my readiness for this position with someone who had experience with the process. Without much consideration, I was told I wasn't ready. Instead, they started discussing other faculty members who were better suited, who happened to be male, for promotion. This very much aligns with an article from the AAMC: women are promoted later compared to men and often receive less sponsorship.

7. Do you have any advice for other women in the field?
Find your own voice. What I mean by that is that everyone has a different way of managing difficult situations at work. You can learn tips on how to handle confrontations, but how you actually handle them and what you actually say has to be your authentic self. It has to be natural to you.

Other advice is to support each other. I have often heard that other women are the reason that women are not promoted or given opportunities. We should be better sponsors if we want to improve inclusiveness.

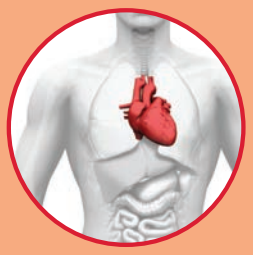
8. How do you balance work and personal life?
I am very lucky to have a spouse who is supportive. He understands that my schedule is unpredictable. There are many evenings I have meetings or the operating room runs late. There is never any judgement or criticism. Also, sometimes you just have to say enough is enough. And go home.

9. What is something you enjoy doing outside of work?
WWe have a large kitchen at our house. I love to cook and bake. We entertain when we can. Food is a large part of our lives. We love trying new restaurants, then trying to make the dishes at home. Similar to medicine being an art form, cooking is also an art form. The fun is trying to follow recipes, then trying to change the recipe, or trying to make something you ate once and seeing how it comes out!

10. Would you change anything about the path you took to get to where you are now?
None of us will ever know how our lives would have ever turned out if we had taken a different path. Each of our experiences shape us, affect us, and make us who we are. I am not sure if I had taken a different path if I would have ended up where I am. I do not have any regrets or wish for a different path.

11. What was the best piece of advice you received?
When I was a medical student, a faculty member had told me you know when you have found the right career when you are excited to go to work. You know it when you enjoy your day at work and find fulfillment even after the most boring of days.

I am lucky that I still enjoy most (!) days at work. I still find clinical work interesting. I also enjoy the challenges of the educational and leadership aspects as well.



ECHO CORNER

TEE EVALUATION OF TV INSUFFICIENCY (TR)

LEARNER NOTIFICATION
Society of Cardiovascular Anesthesiologists
Activity Title: 2025 Echo Corner (TEE Evaluation of TV Insufficiency (TR))
Release Date: 6/2/2025
Expiration Date: 6/2/2027
Activity Type: Enduring Material

Acknowledgement of Financial Commercial Support
No commercial support was received for this educational activity.

Acknowledgement of In-Kind Support
No in-kind support was received for this educational activity.

Accreditation Statement
The Society of Cardiovascular Anesthesiologists is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

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

Description:
The mission of the SCA Newsletter Sub-Committee is to inform the membership of the activities of SCA. The goal of the SCA Newsletter Sub-Committee is to produce and distribute the SCA official newsletter, the SCA Newsletter, six times per year. Each issue of the SCA Newsletter publishes education material including ECHO Corner. ECHO corner cases focus on clinical case presentation of diverse echocardiographic diagnosis encountered in clinical practice relevant to cardiothoracic anesthesiologists.

Educational Information

Physician Practice Gap:
Echo Corner of the SCA newsletter is a written clinical case presentation with echocardiographic images and videos followed by multiple choice questions with explanations. The ECHO corner case review focuses on detailed and concise presentation of clinical findings accompanied by findings on transesophageal echocardiographic (TEE) exam to support the clinical diagnosis. The cases include a written portion with case description, TEE images, and TEE video clips. Three to five multiple choice question are presented to discuss the case. Each question provides an explanation of answer choices and includes a brief discussion of the topic present in each case.

Needs that Underlie the Gap
There is a need to provide education to clinicians on how to perform echocardiographic assessment of the TV, including qualitative and quantitative assessment of tricuspid valve anatomy and function needed to guide surgical or percutaneous interventions.

DESIGNED to Change/Outcome:
Note that in the field of intraoperative echocardiography in general improvements in patient outcomes are difficult to measure because most of the examinations are diagnostic and not therapeutic which are more determinative of outcomes.

SOCIETY OF CARDIOVASCULAR ANESTHESIOLOGISTS
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ECHO CME

Educational Objectives

After completing this activity, the participant should be better able to:

- Identify the anatomic components of the tricuspid valve, including leaflets (anterior, posterior, and septal), annulus, chordae tendineae, and papillary muscle, including normal anatomic variants.
- Determine severity of TR regurgitant jet location, and mechanism of insufficiency (primary vs. secondary).
- Evaluate tricuspid valve anatomy and function preoperatively to guide surgical or percutaneous intervention decisions, including need for intervention at the time of left-sided heart surgery.

Satisfactory Completion

Learners must complete an evaluation form to receive a certificate of completion. Partial credit of individual sessions is not available.

Contact Information

If you have questions regarding your CME certificate, please contact **Natalie Baus at nbaus@veritasamc.com.**

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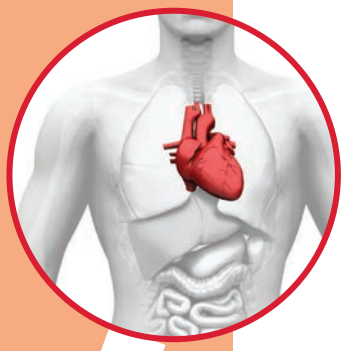

ECHO CORNER

ECHO CASE:
TEE Evaluation of TV Insufficiency (TR)

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CASE PRESENTATION

A 72-year-old female with past medical history of chronic heart failure with preserved left ventricular ejection fraction (LVEF), atrial fibrillation and severe mitral regurgitation presents for mitral valve replacement. Transesophageal echocardiogram (TEE) prior to cardiopulmonary bypass is shown below.

WATCH VIDEO 1 WATCH VIDEO 2

1. **Which of the following TEE imaging levels are used to evaluate tricuspid valve (TV) anatomy and function?**

   A) Mid-esophageal imaging level
   B) Deep esophageal imaging level
   C) Transgastric imaging level
   D) Deep transgastric imaging level
   E) All of the above

The following 3D TEE images are obtained during the exam.

WATCH VIDEO 3 WATCH VIDEO 4

2. **Which of the following statements most accurately describes anatomical relationships of the tricuspid valve and its surrounding structures?**

   A) The tricuspid valve orifice is circular and lies perpendicular to the interventricular septum, with uniform leaflet size and papillary muscle attachments
   B) The anteroseptal commissure is adjacent to the coronary sinus ostium and is best visualized in the ME views
   C) The septal leaflet is fixed to the interventricular septum via multiple chordae and lies inferior and posterior to the aortic valve annulus.
   D) The tricuspid annulus is rigid and saddle-shaped, maintaining a constant shape throughout the cardiac cycle, optimizing coaptation.

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TEE evaluation reveals trileaflet TV with functional tricuspid regurgitation.

3. **Which of the following mechanisms is most commonly associated with tricuspid regurgitation?**

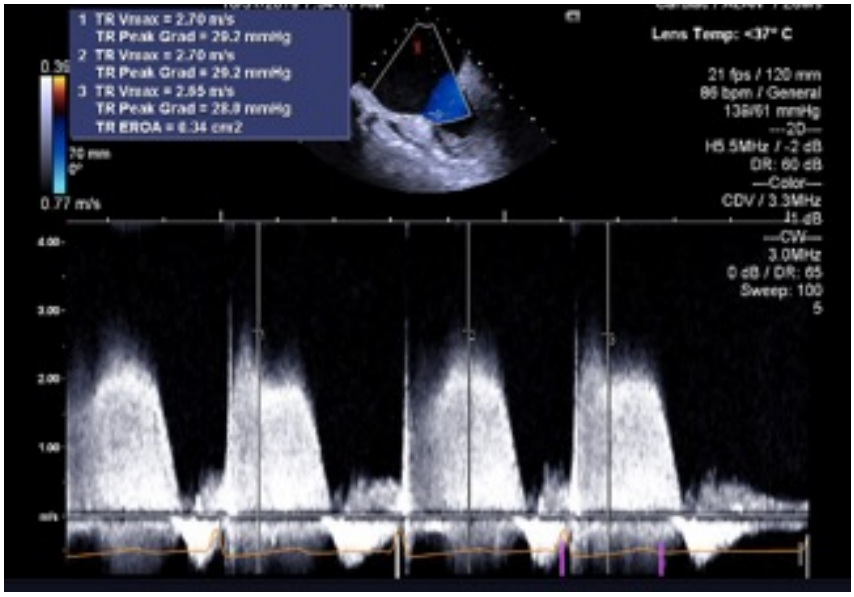
   A) Thickening and retraction of the tricuspid valve leaflets due to rheumatic heart disease

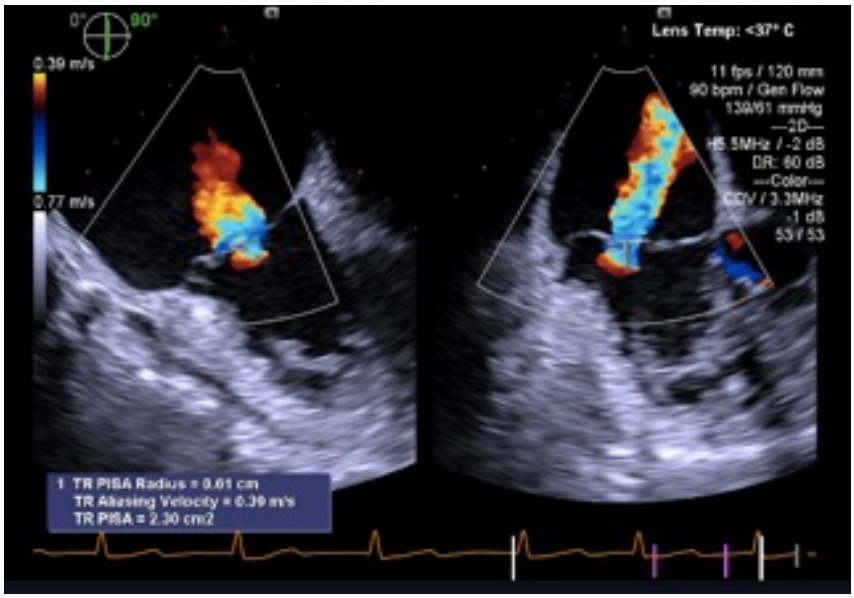
   B) Annular dilation leading to poor leaflet coaptation due to right ventricular or right atrial enlargement

   C) Structural defect in the tricuspid valve cusps resulting from infective endocarditis

   D) A congenital malformation causing displacement of the tricuspid valve, as seen in Ebstein's anomaly

TV Doppler evaluation reveals PISA radius 0.61cm, dense CWD regurgitant jet, EROA 0.34 cm2, and TV annular diastolic dimension of 4 cm.





4. **Which of the following vena contracta widths is most consistent with severe tricuspid regurgitation?**
   A) < 0.3 cm
   B) 0.4–0.6 cm
   C) ≥ 0.7 cm
   D) 0.3–0.5 cm

TR severity is graded as moderate in presence of TV annular dilation.

5. **What is a Class I recommendation for TV valve intervention?**
   A) Isolated TV surgery in asymptomatic severe TR
   B) Surgery for severe TR at the time of left sided surgery
   C) Re-op for isolated TV surgery for symptomatic TR with prior left-sided surgery
   D) Isolated TV surgery for symptomatic primary TR with or without remodeling

In this patient, tricuspid annular diastolic diameter >40 mm indicates the risk of progressive or persistent TR after other surgeries. In such cases, TV surgery is needed and these findings definitely warrant discussion with the surgical team. Based on the current management guidelines, this patient underwent TV annuloplasty at the time of mitral valve surgery.

ANSWERS

Question 1:

Answer: E - All of the above

The aim of TEE evaluation of TV is to quantify TR severity and mechanism, determine leaflet morphology and anatomic variations, assess annular anatomy, and suitability for percutaneous valve interventions. TEE imaging of the TV is anatomically challenging due to its anterior location, large orifice area requiring a wide field of view, and thin leaflets. Furthermore, imaging relies on low quality lateral resolution due to non-perpendicular alignment of annular plane. To overcome these challenges, several TEE imaging levels are required for a comprehensive exam. In addition to mid-esophageal (ME), transgastric (TG), and deep transgastric (DT), recent guidelines suggest deep esophageal (DE) imaging plane (Figure 1). Two imaging planes at ME level include ME four- chamber view at 0 degrees and the ME V inflow-outflow view at 60 degrees. TG imaging level is ideal for identifying leaflet and subvalvular morphology. DT views are obtained by advancing TEE further into stomach with anterior flexion. This imaging level allows ultrasound beam alignment with flow across the TV permitting accurate Doppler interrogation. Finally, to obtain the DE imaging plane, the TEE probe is inserted into the distal esophagus immediately superior to the diaphragm, yielding a view of the right atrium (RA), coronary sinus, and RV with orthogonal view imaging of the right ventricular outflow tract. Comprehensive planar, volumetric, and functional imaging should be obtained at each imaging level.

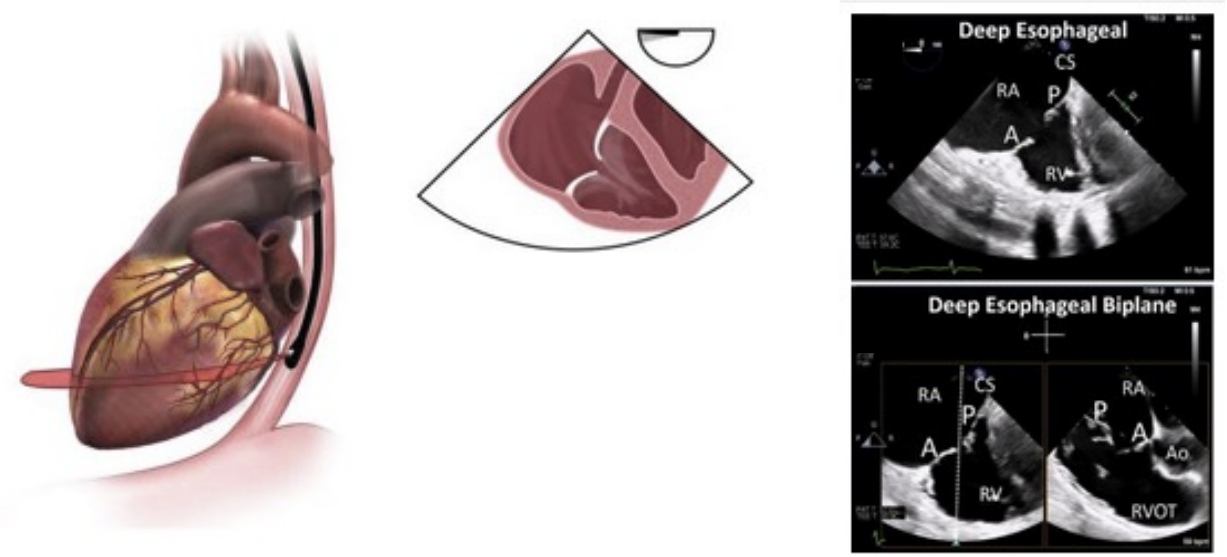


Figure 1. Deep esophageal (DE) imaging plane is obtained by advancing TEE probe into distal esophagus, brining probe close to TV annulus. Simultaneous biplane imaging allows for comprehensive TV exam. RA – right atrium, A – anterior TV leaflet, P – posterior TV leaflet, CS- coronary sinus, RV – right ventricle, Ao -aortic valve, RVOT – right ventricular outflow tract. Modified from Hahn RT, Saric M, Faletra FF, et al. Recommended Standards for the Performance of Transesophageal Echocardiographic Screening for Structural Heart Intervention: From the American Society of Echocardiography. *J Am Soc Echocardiogr.* 2022 Jan;35(1):1-76.

Question 2:

Answer: C - The septal leaflet is fixed to the interventricular septum via multiple chordae and lies inferior and posterior to the aortic valve annulus.

The TV apparatus is a dynamic structure that is more anatomically variable than the left-sided valves. The normal tricuspid annulus has a complex elliptical nonplanar shape. It is about 20% larger and less symmetric than the ''saddled-shaped'' mitral annulus. The classic TV anatomy comprises of 3 leaflets: septal, anterior and posterior. However, there is significant heterogeneity of leaflet configuration and number. Four morphological types have been described: three leaflet (54%), four leaflet (39%), two leaflet (5%), and five leaflet (2%) (Figure 2). To identify them correctly several structures need to be identified: anteroseptal commissure, interventricular or interatrial septum, and anterior papillary muscle. The septal leaflet is the smallest and is associated with interatrial septum. The anterior leaflet extends from anteroseptal commissure to the anterior papillary muscle and is the largest and most mobile of the leaflets. The posterior leaflet extends from anterior papillary muscle to the posterior right ventricular wall. In contrast to mitral valve, there is no accepted standardized imaging display for 3D en face view of TV. Although a standardized en face TV display has been previously suggested in the 2012 EAE/ASE recommendations for 3D echocardiography, the most recent recommendations for structural heart interventions suggest a different orientation because en face surgeon's view (Figure 3), similar to the MV en face view, requires a 180 degree rotation. Lack of a standardized 3D image orientation introduces room for error when identifying TV structures, especially because there is variability in the number of TV leaflets. To avoid errors, the consistent identification of leaflets relies on identification of surrounding landmarks (coronary sinus, interatrial septum, aortic valve), which should be included in the imaging display.

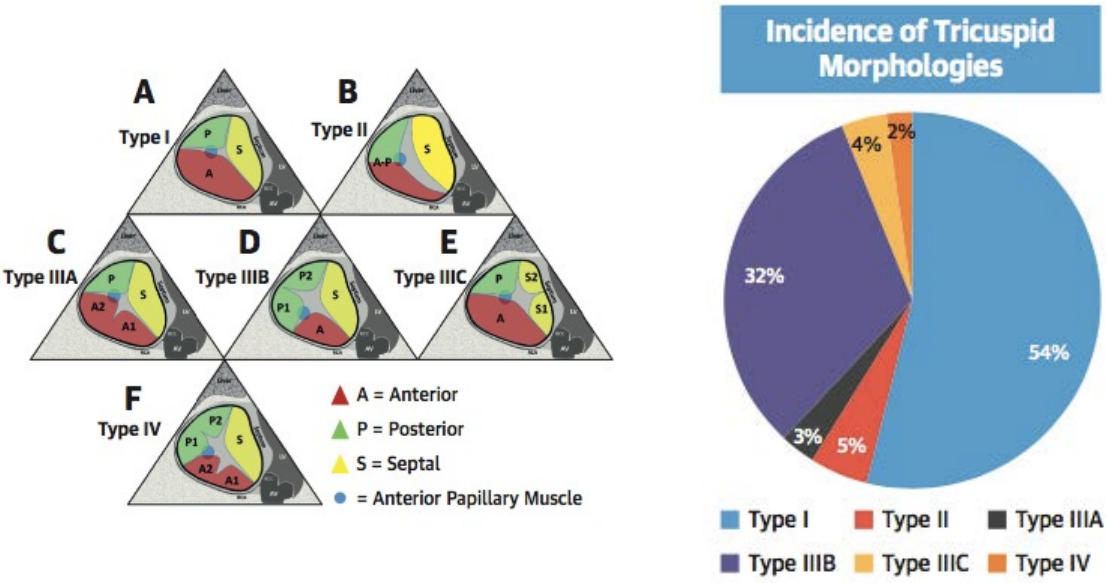


Figure 2. Variability of TV morpholoy. Adapted from Hahn RT, Thomas JD, Khalique OK, Cavalcante JL, Praz F, Zoghbi WA. Imaging Assessment of Tricuspid Regurgitation Severity. JACC Cardiovasc Imaging. 2019 Mar;12(3):469-490.

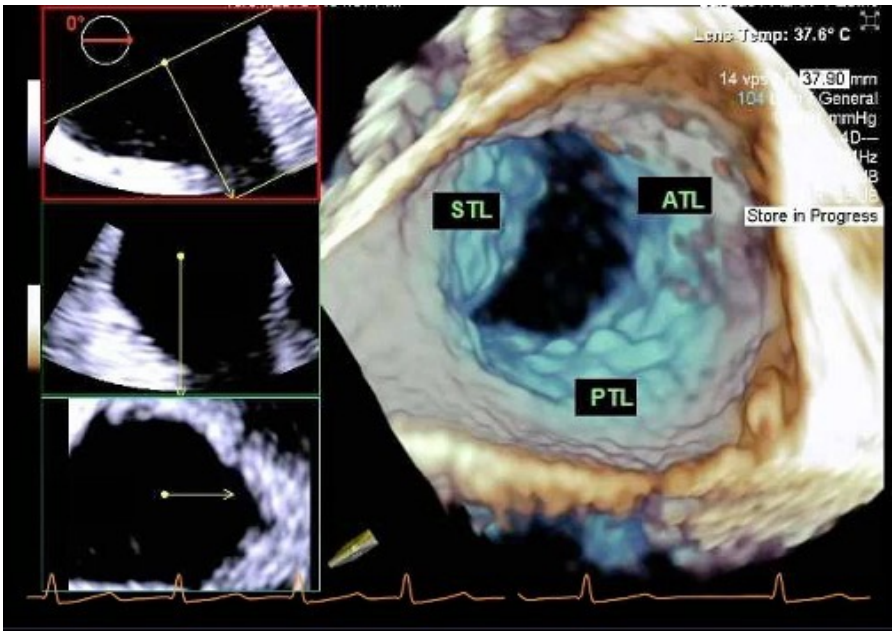


Figure 3. 3D en face view of Tricuspid Valve. STL – septal leaflet, ATL – anterior leaflet, PTL – posterior leaflet

Question 3:

Answer: B - Annular dilation leading to poor leaflet coaptation due to right ventricular or right atrial enlargement

There are multiple causes of TR that vary in their pathological mechanism and affect decisions about treatment. The etiology of >90% of TR is secondary/functional and is due to right ventricular (RV) and right atrial (RA) remodeling in the setting of tricuspid annular dilation (with or without leaflet tethering) that can be resulting from left-sided valvular or myocardial disease, pulmonary hypertension, chronic RV pacing with induced dyssynchrony, atrial arrhythmias (atrial fibrillation) with RA enlargement and annular remodeling. Irrespective of the specific initial etiology, TR is a progressive disease in the setting of RV and RA remodeling. Functional TR worsens over time and despite goal oriented medical therapy, the natural history of persistent TR is unfavorable. The remainder of TR is due to primary etiology, including rheumatic heart disease, endocarditis, carcinoid, drug toxicity, radiation, and congenital heart disease.

Question 4:

Answer: C - ≥ 0.7 cm

American society of echocardiography guidelines focus on qualitative, semiqualitative, and quantitative assessment of TR. Integration of multiple parameters is required to accurately grade TR severity because no single Doppler or echocardiographic measurement or parameter is precise enough. Qualitative Doppler parameters include color flow area, flow convergence zone, and continuous flow doppler jet. Semiqualitative parameters include color flow jet area, vena contracta width, PISA radius, hepatic vein flow, and tricuspid flow. Finally, quantitative paraments include EROA and regurgitant volume. When multiple parameters are concordant, TR grade can be determined with high probability (Figure 4). Of note, a new dedicated grading scheme for TR has been proposed with 5 grades of TR severity: mild, moderate, severe, massive, and torrential.

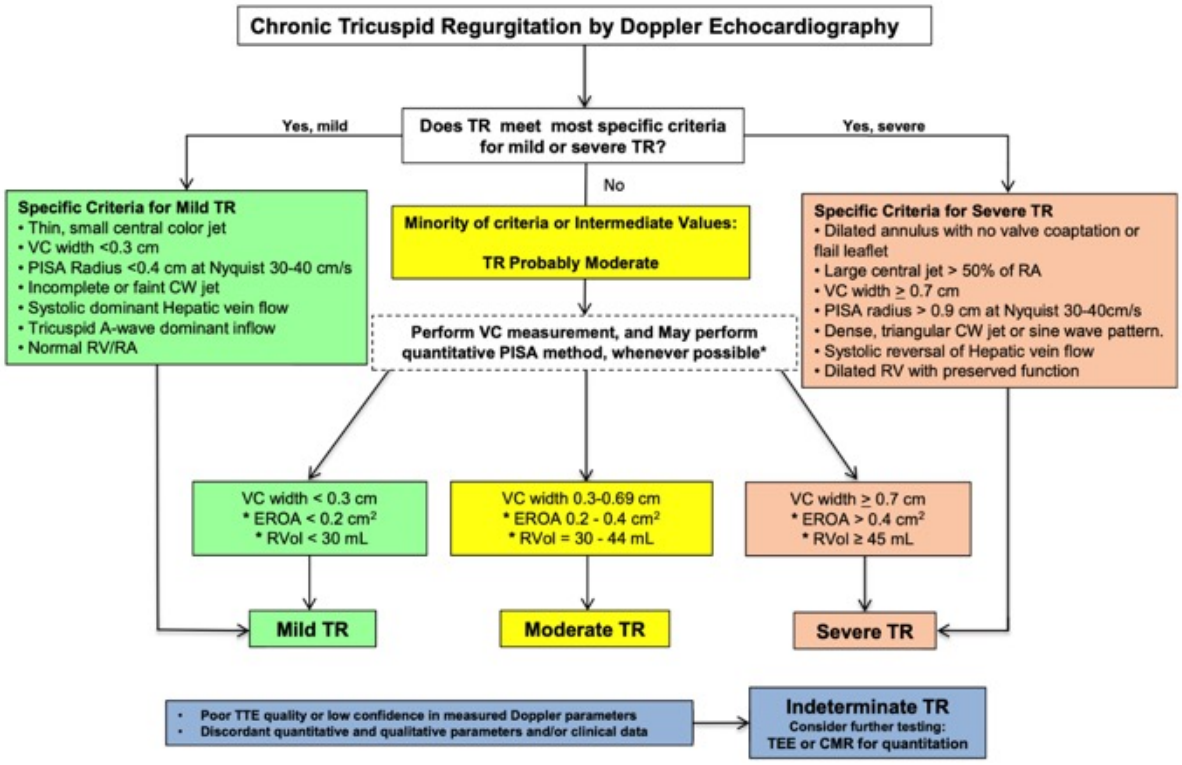


Figure 4. Algorithm for the interpretation of multiple parameters of TR severity. Adopted from Zoghbi WA, et al. Recommendations for noninvasive evaluation of native valvular regurgitation: a report from the American Society of Echocardiography developed in collaboration with the Society for Cardiovascular Magnetic Resonance. J Am Soc Echocardiogr. (2017) 30:303–71.

Question 5:

Answer: B - Surgery for severe TR at the time of left sided surgery

Based on the ACC/AHA guidelines for treatment of valvular disease, currently the only class I recommendation is in patients with severe TR undergoing left-sided valve surgery. In patients with progressive TR undergoing left-sided valve surgery, TV surgery can be beneficial in context of either 1) tricuspid annular dilation or 2) signs and symptoms of right-sided heart failure (HF) (Class II recommendation). Surgical treatment is performed for selected patients with TR at the time of surgery for left-sided valve lesions to treat severe TR and to prevent later development of severe TR in patients with progressive TR. In patients with signs and symptoms of right-sided heart failure and severe primary TR, isolated TV surgery can be beneficial to reduce symptoms and recurrent hospitalizations (Class II recommendation). In patients with signs and symptoms of right-sided HF and severe TR who have undergone prior left-sided valve surgery; reop for isolated TV surgery may be considered in the absence of severe pulmonary hypertension or severe RV dysfunction (Class II recommendation).

Class of Recommendation	Level of Evidence	Recommendation	Risk vs Benefit
1 recommended	B-NR	Surgery for severe TR at the time of left sided surgery	Benefit >>>>Risk
2a reasonable	B-NR	Surgery for progressive TR at the time of left-sided surgery, if remodeling is present	Benefit >>Risk
2a reasonable	B-NR	Isolated TV surgery for symptomatic primary TR with or without remodeling	Benefit >>Risk
2b may be considered	B-NR	Isolated TV surgery in asymptomatic severe TR	Benefit ≥ Risk
2b may be considered	B-NR	Re-op for isolated TV surgery for symptomatic TR with prior left-sided surgery	Benefit ≥ Risk

Figure 5. Recommendations for TV intervention. Adapted from Otto CM, Nishimura RA, Bonow RO, Carabello BA, Erwin JP 3rd, Gentile F, Jneid H, Krieger EV, Mack M, McLeod C, O'Gara PT, Rigolin VH, Sundt TM 3rd, Thompson A, Toly C. 2020 ACC/AHA Guideline for the Management of Patients With Valvular Heart Disease: Executive Summary: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2021 Feb 2;143(5):e35-e71.

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Right Ventricle Functional Echocardiographic Cutoffs in Patients with Compared to Without Tricuspid Regurgitation

J Am Soc Echocardiogr. 2025 Mar;38(3):228-235. doi: 10.1016/j.echo.2024.10.012. Epub 2024 Oct 30. PMID: 39486691 / Zornitzki L, Freund O, Frydman S, Rozenbaum Z, Granot Y, Banai S, Topilsky Y. Mortality-Based

Reviewer:

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Department of Anesthesiology
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Over the last 3-4 decades the awareness of the right heart and the methods to evaluate function have increased. Arguably the ability of echocardiography to perform bedside non-invasive assessment of heart function plays a critical role in patient management. 'Eyeballing' or qualitative assessment of right ventricular (RV) size and systolic contractility is not well supported.[1,2] Although quantitative assessment of RV size and function with 2D and 3D echocardiographic is feasible, limitations include the complexity of the RV shape/anatomy, imaging planes and angles, imaging quality, and accurate endocardial detection.[2]

Longitudinal contraction (base (tricuspid valve annulus) to RV apex) is thought to represent 60-80% of ventricular contractile performance.[3,4,5,6,7] Tricuspid annular plane systolic excursion (TAPSE) and tissue Doppler imaging (TDI) measurement of the peak tricuspid annular systolic velocity (S') are two relatively simple assessments of longitudinal contraction. During TTE, TAPSE and S' are measured from the apical four chamber window with the cursor placed along the longitudinal axis from the apex through the lateral tricuspid valve annulus. TAPSE is measured using m-mode echocardiography and S' is measured during pulse wave tissue Doppler.[1,2]

Based on multiple reports including thousands of patients without clinical cardiovascular disease published guidelines report that a TAPSE < 17mm or an S' < 9.5 cm/s suggests the presence of RV systolic dysfunction.[1,2,8] Although these lower inflection points are written, the data for patients without clinical cardiovascular dysfunction are spread over a wide range. For these patients without cardiovascular dysfunction TAPSE ranges from 15-31mm with a mean of 23-25 mm.[2,9,10,11] Guzmán-Sánchez reported that 2.9xTAPSE would yield RVEF measured by cardiac MRI.[11] Lopez-Candales reported that a TAPSE of < 20mm and an RVFAC < 40.9% indicating dysfunction.[12] Normal tissue annular systolic velocity (S') ranges from 9-20cm/s with a mean of 15 cm/s.[2] Mor-Avi reported that TDI S' < 11.5cm/s is suggestive of dysfunction, and < 9.5cm/s is predictive of adverse outcome.[13] Meluzin et al concluded that an S' < 11.5cm/s was associated with an RVEF (nuclear imaging) < 45%.[14]

ARTICLE: Zornitzki et al (16)

Zornitzki et al retrospectively examined echocardiographic data, with specific emphasis on the right ventricle (RV), and their association with mortality.[15] They had specific interest on the Tricuspid Annular Plane Systolic Excursion (TAPSE) and the peak lateral tricuspid annular systolic velocity (S'). The results were reported for the entire population and then stratified for the presence of moderate or greater tricuspid regurgitation determined according to guidelines.[16]

24,717 were included in the analysis. Patients were followed for an average of 1,321 days. Approximately 5% (1,143) patients had moderate or greater TR. Mortality data were analyzed using Kaplan-Meier method. Univariate and multivariate models were used to calculate hazard ratios (HR). The data were displayed using spline curves with hazard ratios on the y-axis and the RV parameter on the x-axis. An HR > 1 was considered excess mortality.

For the entire cohort a TAPSE < 20.9 mm, an S' < 10.9cm/s, and, interestingly, a S' > 20.0cm/s were associated with excess mortality. For patients with moderate or greater TR, a TAPSE

< 18.0mm, and an S'< 10.0 were found to be significant predictors of mortality. Further stratification based on severe (vs non-severe) TR yielded greater mortality when TAPSE < 17.7mm and S'< 9cm/s were associated with excess mortality. For those without significant TR a TAPSE < 21.5mm and S' < 10.9cm/s or > 20.0cm/s were significant associated with mortality.

Multivariate analysis reported that a TAPSE < 20.9 was associated with higher mortality. When an analysis was performed without TAPSE, S' < 10.9cm/s was an independent predictor of mortality. The exclusion of cardiac surgical patients did not alter the TAPSE and S' thresholds. Other multivariate factors associated with higher mortality included severe TR, RV dilation, pulmonary artery systolic pressure, left ventricular ejection fraction, age, gender, elevated lipids, diabetes, heart failure, and lung disease.

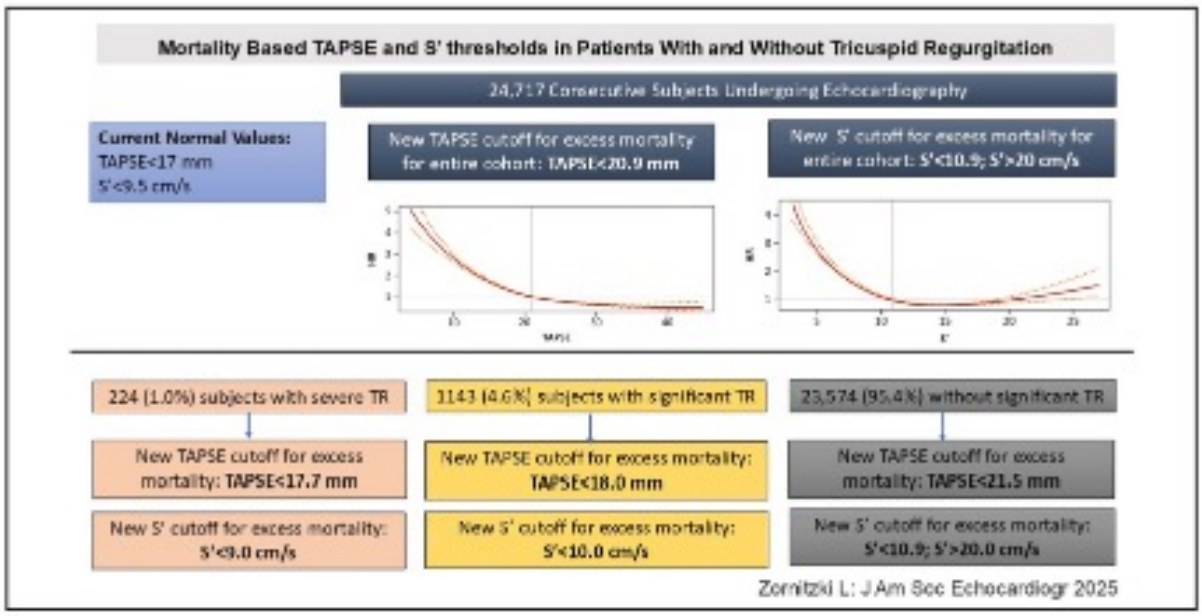
Table 1 and supplemental Table S1 compare TAPSE <20.9 to > 20.9mm and compare S'<10.9 to > 10.9cm/s respectively. Without going into details, patients with lower TAPSE and S' and greater comorbidities, greater left heart systolic and diastolic dysfunction, greater left heart valve dysfunction, and reduced stroke volumes and cardiac output/index. Patients with lower TAPSE and S' also have greater occurrence of moderate or more tricuspid regurgitation, and moderate or greater dilated RV size.

An interesting finding was that S' > 20cm/s were also associated with greater mortality. The authors speculated that the higher S' reflected other systemic illness that could be associated with a 'hyperadrenergic state', which might be associated with greater mortality.

For patients with significant TR, excess mortality was associated with lower TAPSE and S' for patients. This was unexpected as the authors thought that, like mitral regurgitation, a reduced ventricular afterload would increase longitudinal contraction. In further analysis, ventricular TR (associated with pulmonary hypertension) which represented higher afterload, and atrial TR (atrial fibrillation, atrial dilation, RV dilation) which represented higher preload were analyzed. Compared to atrial TR, ventricular TR recorded lower TAPSE (17.6mm vs 20.5mm) and S' (9.84cm/s vs 10.5cm/s). Both of these are lower than the whole population and more so than those without significant TR. The authors speculated that ventricular TR (i.e. greater afterload) was associated with greater contractile dysfunction and atrial TR (i.e. greater preload) was associated with larger chamber volumes impacting on myofibril direction.

Finally, the authors analyzed the impact of lower TAPSE and higher SPAP and found that not only were both significantly associated with greater mortality, but the combination of TAPSE/SPAP was a greater predictor than either of the two variables alone.

The authors concluded that TAPSE and S' thresholds of 20.9mm and 10.9cm/s respectively were associated with excess mortality.[15] Patients with significant and severe TR have lower thresholds including a TAPSE of 17-18 mm and S' 9-10cm/s. The authors refer to these thresholds as 'new cutoff' while discussing the currently described cutoffs (TAPSE < 17mm and S' < 9.5cm/s) included in guideline papers.[1,2]



Commentary

Based on healthy patients, the current guidelines describe a TAPSE of < 17mm and an S' < 9.5cm/s suggestive of RV dysfunction.[1,8] These guidelines are based on a number of articles describing the association between these measures of longitudinal contractile function and reference measures of RV function and outcome.[1,8,17] It isn't clear that the guidelines need to be rewritten based on the results of the study by Zoritzki et al since they are not describing the same end point i.e. RV function vs mortality.[1,2,15] To add, the guidelines are reporting the lower inflection points, which does not reflect the range of findings for a population without clinical cardiovascular dysfunction.[2,9,10,12,14] The thresholds associated with mortality described by Zonitzki are not too different than that found in the literature to be associated with reduced RV function.

A further look at the article by Zornitzki et al, including tables 1, 2, and supplemental tables allude to other explanations for the 'excess mortality'.[15] Tables 1 and Supplemental Table 1 (S1) compare demographics and cardiovascular assessments based cutoffs for TAPSE = 20.9mm and S' = 10.9cm/s. Those below these cutoffs have significantly greater co-morbidities, significantly greater left heart valve dysfunction, and greater left heart systolic and diastolic dysfunctions.[15] Patients with lower TAPSE and S' also had higher pulmonary pressures. Perhaps the reported thresholds are reflective of hemodynamic impact of left heart dysfunction and the mortality is, to a large part, due to comorbidities and left heart dysfunction.

There were no other measurements of RV contractility.[15] While the range of RAP was greater with lower TAPSE and S', the mean RAP was the same (5mmHg) suggesting that these specific thresholds were not reflective of venous congestion i.e. clinically apparent right heart failure. A dilated RV or moderate or greater TR was found in 9-10% of patients with a TAPSE < 20.9mm or S'< 10.9cm/s vs 2% of those with TAPSE > 20.9 or S > 10.9cm/s. There was no analysis comparing those with dilated RV or those with moderate or greater TR to those without.

Relying solely on TAPSE or S' to determine RV contractility is problematic. The right ventricle contracts in three directions; radial, circumferentially, and longitudinally. Basing care and outcome on longitudinal contractile measures is limited as it relies on a 'single segment' measure in determining the function of a three dimensional structure.[4,6,18] Although prior data report that longitudinal contraction represents 60-80% of ventricular contractile performance, other data vary as to the distribution of contractile forces with longitudinal contribution ranging from 20-80%, being affected by cardiovascular geometry and function.[3,4,5,6,7]

Prior investigation show that TAPSE and S' are not synonymous with RV performance which is

better defined hemodynamically by RV pressure generation (RV dP/dt) and pulmonary artery flow (QPA).[19,20,21] Sixteen patients with chronic thromboembolic pulmonary hypertension (CTEPH) were evaluated pre and post pulmonary endarterectomy (PEA). While RV ejection fraction and S' improved with afterload reduction, TAPSE decreased from 14.5mm prior to surgery to 8.5mm at 1 week and 11mm at 6 months.[20] The authors speculated that this was due to alterations in contractile patterns and apical motion and rotation.[20] Bootsma et al reported reductions in TAPSE and S' upon sternotomy and pericardiotomy for patients undergoing cardiac surgery.[19] TAPSE declined from 20.9 to 9.1mm and S' declined from 8.7 to 7.2 cm/s, while myocardial performance index, RV ejection fraction (RVEF), and hemodynamic performance did not change, or even improved.[19] Bootsma et al described that sternotomy and pericardiotomy alters RV size, geometry, and myocardial fiber direction resulting in a reduction in longitudinally running fibers which was compensated with an increase in circumferentially directed fibers and contraction.[19] In both heart transplant and CABG patients a post bypass decline in longitudinal function did not equate to improved RV performance and hemodynamic stability.[7] On the other hand, a report of patients with pulmonary hypertension, TAPSE overestimated RV function.[18] The authors concluded that for patients with PHTN, changes in geometry and contractile direction resulted in 'clockwise rotation' due to left heart compression, overestimating right heart function.[18]

Both the presence and the causes of TR can result in a dilated and dysfunctional RV, which is associated with higher mortality. In the study above, when comparing mild or less TR to those with significant TR, excess mortality was found with lower TAPSE and S'. Hsiao et al also reported lower TAPSE and S' with greater TR.[22] In this article, RVEF quantified by echo and radionuclide imaging exam were similar.[22] The association between RVEF and longitudinal measures declined from moderate in cases of mild TR to poor in patients with severe TR.[22] Perhaps the lower longitudinal are more reflective of geometrical changes and myofibril orientation than they are of RV function and/or predictors of mortality.

The data from the present study and all others include measurement error. The results report missing data in thousands of cases. Measurement errors should be considered with regard to the accuracy and/or predictability of TAPSE and S'.[23,24] M-Mode and Doppler analysis are affected by scan line alignment (angle) with tissue movement, radial and circumferential movement in addition to longitudinal movement, imaging difficulty, and operator error. On average 10% is expected for 2D and m-mode analysis.[23,24] Without proper alignment between imaging planes and annular motion the error rate may be > 7%.

The finding that TAPSE/PASP was a more significant predictor of mortality than either TAPSE or PASP was stated but not emphasized. The ratio has previously been described as a bedside measure of RV-PA coupling, a concept that is the basis of hemodynamic performance of the right heart.[25,26] This describes the ability of the right ventricle to generate enough pressure (RV dP/dt) to overcome the resistance of the pulmonary vasculature to generate pulmonary blood flow (QPA).[25,26,27,28,29,30] With regard to patient outcome, RV-PA coupling is critical to hemodynamic stability and associated with patient outcome across different populations.[1,2,25,26,27,28,29,30]

It is not clear that future guideline need to be altered significantly except to include the range of TAPSE and S' associated with 'normal' RV function and mortality. The information gained from TAPSE and S' are important components of RV functional assessment, however, their meaning should be considered as part of a bigger picture. Perhaps guidelines should encourage more quantitative assessment of RV size, ejection fraction, and measures of coupling to improve assessment of hemodynamic performance

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Intraoperative High and Low Blood Pressures Are Not Associated with Delirium After Cardiac Surgery: A Retrospective Cohort Study

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Objective
This retrospective cohort study aimed to evaluate whether intraoperative hypotension (<60 mmHg) or hypertension (>80 mmHg) during different phases of cardiac surgery (before, during, after cardiopulmonary bypass) is associated with postoperative delirium (POD).

Key Findings
Sample: 11,382 adult patients undergoing cardiac surgery with CPB at Cleveland Clinic.

Primary Outcome
Incidence of POD within 12–96 hours post-op, assessed via CAM, CAM-ICU, and bCAM tools.

Main Result
No statistically or clinically significant association between TWA-MAP <60 mmHg or >80 mmHg and POD at any intraoperative phase. Delirium Rate: 6.0% (678 patients).

Strengths
Large Cohort Size: Enhances statistical power and generalizability within the cardiac surgery population.

Granular Intraoperative Data: MAP was recorded every minute with artifact correction and time-weighted averaging for accuracy.

Robust Statistical Modeling: Use of multivariable logistic regression with spline analysis and sensitivity testing for alternative MAP thresholds (<55, <50 mmHg).

Well-Controlled Confounding: Adjusted for multiple perioperative risk factors (age, comorbidities, surgical time, etc.).

Potential under detection of Delirium.

Reliance on routine nursing assessments may have underreported transient or subtle delirium episodes.

The reported POD rate (6%) is lower than many other studies, which often range between 10–30%.

Fixed MAP Thresholds: Uniform MAP cutoffs (<60, >80 mmHg) may overlook individualized cerebral autoregulation thresholds, which vary widely between patients.

Focus Limited to Delirium: While delirium is a relevant cognitive outcome, the study does not address other BP-related complications such as stroke, bleeding, or renal failure.

Limitations
Potential under detection of Delirium:

Interpretation
The study convincingly shows that moderate deviations in intraoperative MAP (within studied ranges) are not independently associated with POD. However, this does not negate the clinical need for tight BP control during cardiac surgery, as other complications beyond delirium (e.g., stroke, hemorrhage) remain critically dependent on hemodynamic precision.

Clinical Implications

Intraoperative hypotension/hypertension may not increase delirium risk in isolation, but blood pressure control remains crucial for avoiding other adverse outcomes.

Supports a more nuanced, individualized BP management approach, potentially using autoregulation-based monitoring.

Findings should not be misinterpreted to support relaxation of BP targets during high-risk procedures.

Conclusion

This high-quality study contributes important data to the debate around intraoperative BP targets and cognitive outcomes. While it refutes a link between MAP extremes and delirium within common surgical ranges, it reinforces the complexity of POD pathophysiology and underscores the need for multifactorial prevention strategies in cardiac surgical patients.

Efficacy of Intravenous Iron Supplementation in Reducing Transfusion Risk Following Cardiac Surgery: An Updated Meta-Analysis of Randomized Controlled Trials

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Background

Anemia, defined by the World Health Organization as hemoglobin <13.0 g/dL in men and <12.0 g/dL in women, is a frequent complication of cardiac surgery, with incidence estimates ranging from 29 to 94%.[1,2] Red blood cell (RBC) transfusion is commonly used to treat postoperative anemia, but is associated with a wide array of adverse postoperative events, including acute kidney injury, infection and mortality.[3] Patients with preoperative anemia are more likely to experience postoperative morbidities and to receive RBC transfusions.[4]

Iron deficiency is a common cause of preoperative anemia.[4,5] Intravenous (IV) iron therapy is a fast and effective means of repleting iron stores.[6] However, its impact on postoperative RBC transfusion risk and other clinical outcomes following cardiac surgery is unclear. The goal of the recent meta-analysis by Hung and colleagues was to evaluate the impact of preoperative IV iron therapy on these outcomes.[7]

Methods

Major databases were searched for randomized controlled trials (RCTs) comparing intravenous iron therapy to oral iron or placebo in cardiac surgery patients.

Studies were eligible for inclusion if they enrolled adult patients with or without preoperative anemia, who underwent any type of cardiac surgery. Intravenous iron could be administered pre-, intra- and/or postoperatively, and in any formulation and in conjunction with other anemia treatments (e.g. erythropoietin), and compared to a control group receiving either oral (PO) iron or placebo.

The primary outcome was the incidence of RBC transfusion after cardiac surgery, up until hospital discharge or 30 days postoperatively. Secondary outcomes included the number of RBC units transfused, postoperative hemoglobin levels (assess on postoperative day [POD] 1, PODs 4 - 10, and POD >21), iron status, postoperative complications (AKI, infection, mortality), and hospital length of stay.

Results

Fourteen randomized controlled trials were identified, which were comprised of 2043 subjects. Patient mean age ranged from 53.2 to 75 years. Male patients represented 35% to 91% of study patients. Mean hemoglobin values ranged from 9.5 to 14.7 g/dL. A variety of IV iron formulations were used.

Intravenous iron therapy significantly reduced RBC transfusion risk (relative risk [RR] 0.77, 95% confidence interval [CI] 0.65 - 0.91, p = 0.002, n = 1955, I2 = 0.61%). Age was the only significant covariate for RBC transfusion risk (coefficient 0.03, p = 0.03), with younger patients more likely to see a larger benefit.

PRB transfusion risk was similar (p = 0.5) when comparing studies that administered only IV iron to those that administered IV iron and erythropoietin together (RR 0.79, 95% CI 0.64 - 0.97, p = 0.03, I2=56%, 10 RCTs, n = 1181 versus RR 0.69, 95% CI 0.48 - 0.99, p = 0.04, I2=77%, 4 RCTs, n = 774).

PRBC transfusion risk was significantly reduced in patients with and without preoperative anemia (RR 0.75, 95% CI 0.58 - 0.95, p = 0.02, I2 = 77%, 8 RCTs, n = 1274 versus RR 0.78, 95% CI 0.66 - .92, p = 0.004, I2 = 0%, 5 RCTs, n = 681). Risk of transfusion across anemic and non-anemic subgroups was similar (p = 0.77).

Postoperative hemoglobin values were significantly higher in the IV iron group on PODs 4 – 10 and POD > 21. Patients treated with IV iron also received fewer PRBC transfusions (MD - 0.57 units, 95% CI: -1.09 to -0.06, p = 0.03, =88%, 8 RCTs, n =1332). IV iron patients had higher serum iron levels postoperatively. No differences in mortality, infection, AKI or postoperative length of stay were observed between IV iron and control groups.

Discussion

This meta-analysis found that IV iron therapy was associated with significantly reduced risk of PRBC transfusion in cardiac surgery patients, with younger age associated with a more pronounced benefit. Subgroup analyses demonstrated similar transfusion risk across groups. These findings are consistent with a prior meta-analysis of IV iron therapy in cardiac surgery patients.

In the current study, IV iron therapy was also associated with a small but significant reduction in volume of PRBC transfusion. Notably, although IV iron has previously been suggested to contribute to risk of infection,[8] no association was found between use of IV iron and postoperative infection. Similarly, no association was found between IV iron infusion and other postoperative complications, including mortality, AKI, and prolonged hospital LOS.

Limitations of the current study include marked heterogeneity in patient populations, iron formulations, dosing regimens, and timing of IV iron administration. The relatively short follow up time of included studies also limits conclusions about longer-term effects of iron therapy.

Strengths of the current study include its large sample size and robust statistical analyses, including sensitivity analyses and the clear identification of outliers and individual papers that drove overall findings.

Conclusion

The meta-analysis by Hung and colleagues suggest that IV iron therapy, compared to placebo and/or PO iron, may be beneficial in reducing postoperative RBC transfusion risk in cardiac surgery patients. Although IV iron repletion was not associated with improvements in other clinical outcomes, this study supports the use of IV iron as a strategy to mitigate PRBC transfusion risk regardless of preoperative anemia status.

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PRO CON

Mechanical Circulatory Support (MCS) for Post-Infarction Ventricular Septal Rupture (VSR) Extracorporeal Membrane Oxygenator (ECMO)

VS.

Temporary Ventricular Assist Device (tVAD; Impella)

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Post-Infarction Ventricular Septal Rupture (PI-VSR) is a morbid condition with a mortality approximately 40% (20-80%).[1-5] Presentation ranges from mild or moderate cardiopulmonary dysfunction to cardiogenic shock. Critical components of management and survival include immediate suspicion, emergent imaging to diagnose and define the defect, and therapies directed toward managing cardiogenic shock. Managing cardiogenic shock should be coupled with reducing progression of the injury (e.g. defect expansion), the latter which includes early non-surgical coronary revascularization when possible, and reduction of cardiac loading conditions and stress.

Cardiogenic shock prior to and especially after repair of PI-VSR is the strongest predictor of outcome, independent of the success of the PI-VSR repair.[1-9] The cause or causes of cardiogenic shock and pulmonary dysfunction are multiple and complicated, including primary left ventricular injury and dysfunction, acute mitral valvular dysfunction, +/- right ventricular failure.[3,5,8] The latter results from the volume and pressure overload through the PI-VSR, with or without primary RV infarct, +/- the acute increases in pulmonary artery pressures. The severity of left to right flow across the VSR is related to both defect size and the pressure gradient between the LV and RV.

While it seems intuitive that immediate closure of the PI-VSR should improve outcome, however, increasing data reports that delays in repair of 7, 14, or even 21 days is associated with greater repair success and reduced mortality.[1,3,4,7,10,11,12] Thus, the immediate care emphasis is placed on managing cardiogenic shock and pulmonary dysfunction and not on early repair.

Benefits of delayed repair:

1. Management of cardiogenic shock and recovery of end organ function
2. Infarct and peri-infarct tissue stabilization, reduction, and remodeling
3. Opportunity to assess coronary anatomy and perform percutaneous coronary intervention
4. Opportunity to obtain greater detail of the PI-VSR and associated pathoanatomy

Risks of delayed repair:

1. Poorly controlled cardiogenic shock
2. Progression and/or expansion of infarct/peri-infarct region
   a. Increased shunt
   b. Greater ventricular dysfunction
   c. Pseudoaneurysm formation
   d. Free wall rupture
3. Complications of therapies implemented to manage cardiogenic shock

To tolerate the delay, hemodynamic support and stability are required. While heavy focus is traditionally focused on increasing forward flow, i.e. cardiac output and systemic perfusion, it is as critical to reduce cardiac load, pressure and wall stress to prevent stretch, expansion and progression of the defect, and to allow infarct and peri-infarct tissue stabilization and

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remodeling.[13] Peak breakdown of necrotic tissue under optimal conditions is completed by day 7 and infarct expansion stabilizes within 28 days.[13] Although complete healing may take months, these remodeling data are consistent with a growing body of literature that describes improved repair success and lower mortality when the VSR repair is delayed beyond 7 days.[1,3,4,7,10,11,12]

The use of mechanical circulatory support (MCS) to manage cardiogenic shock and/or facilitate high risk cardiology procedures has evolved and support for early utilization is increasing.[14] While IABP is still most commonly employed, its use has declined while placement of other higher flow temporary devices (ECMO, Tandem Heart, Impella) have risen.[15,16,17,18] Decision as to which MCS is placed is both institution and patient dependent. Additionally, it is possible to combine MCS devices to either balance out the pros and cons of each MCS, and/or have additive effects on favorable hemodynamics and blood flow (e.g. ECMO and IABP or ECMO and Impella 5.5).[19,20,21] The application of MCS for PI-VSR has similarly evolve to improve management of cardiogenic shock and enabling a delay in repair to facilitate tissue remodeling, support the cardiopulmonary functions.[3,8,15,19,22] After surgery it remains critically important to support hemodynamic functions as well as reduce stress on the surgical repair.[3,17]

Veno-Arterial Extracorporeal Membrane Oxygenation (VA-ECMO) is an essentially cardiopulmonary bypass without the reservoir, suction, and cardioplegia delivery system. It assumes the role of the heart and lungs and can delivery a cardiac output of approximately 6L/min. While cannulae can be placed centrally or peripherally, the latter being more common and includes a 50-60 cm x 19-25 Fr cannula placed in the femoral vein advanced into the right atrium, possibly with the tip in the superior vena cava, and a 15-20 cm x 15-25 Fr cannula placed in the femoral arterial with its tip lying in the iliac artery or lower aorta. When placed centrally via an open chest the venous cannula may be > 30 French inserted via the right atrial appendage with its tip in the right atrium. The motor pump is a centrifugal flow pump that is sensitive to afterload.

The Impella MCS is a single cannula (11-23Fr) that can either be placed via the femoral or axillary artery, the latter delivering a systemic flow of up to 5.5 L/min. The tip, or inflow, is advanced across the aortic valve and positioned 4.5-5.5cm into the left ventricle toward the apex. It is an axial flow device that draws blood into the inflow and delivers it just beyond the aortic valve. Femoral cannulae may be placed instead with the device placed similarly as the axillary cannulae. These can deliver flows from 2.5 to 5.0 L/min.

Suffice it to say, each MCS system and placement of cannulae has inherent complications associated with placement of cannulae and coagulation management, including limb or end organ ischemia (Gut; Kidney), thromboembolic complications, bleeding, hemolysis, and stroke.[23,24,25] While IABP may have fewer complications (up to 2.6%) and an associated mortality of 0.5%, it hemodynamic impact is significantly less than VA-ECMO or Impella.[23,24,25] By contrast, VA-ECMO and Impella 5 or 5.5 are associated with significantly large rate of complications including limb ischemia up to 10% and bleeding complications as high as 40-50%, and stroke up to 10%.[23,24,25]

	Intra-aortic balloon pump	Impella	TandemHeart	Veno-arterial ECMO	Central ventricular assist device
Pump mechanism	Pneumatic	Axial	Axial	Centrifugal	Centrifugal
Insertion technique	Descending aorta via femoral artery	Into left ventricle retrogradely via femoral artery and through aortic valve	Cannula into left atrium. Inserted through femoral vein with trans-septal puncture	Peripheral (e.g., femoral artery and vein cannulation) or central (right atrial and ascending aorta cannulation)	Central cannulation (left atrial and ascending aorta)
Risk of limb ischaemia	+	++	++	+++ (if peripheral)	–
Anticoagulation	+	++	++	+++	+++
Haemolysis	+	++	++	++	++

With permission from: Ali JM, Abu-Omar Y. Complications associated with mechanical circulatory support. Ann Transl Med. 2020 Jul;8(13):835. doi: 10.21037/atm.2020.03.152. PMID: 32793680; PMCID: PMC7396259.

MCS devices have benefits and limitations which require continuous patient monitoring to adjust care as needed. Considering that each device has Pro/Con and contraindications, the selection of an MCS device is individualized per the needs of the patient (18). Less common, based on the individual case upgrading the MCS device may be necessary to either add a right sided VAD, or combine different MCS, e.g. IABP and VA-ECMO, VA-ECMO with Impella, or BIPella (18,27). While the hemodynamic benefits of combined MCS devices is clear (28), the risk of device related complications increases significantly (24).

Each MCS option has their 'Pros and Cons' including availability and speed of application, hemodynamics effects, complications, and costs (29). The following Pro/Pro debates whether VA-ECMO or tVAD (Impella 5.5; Abiomed) is the preferred MCS for PI-VSR.

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Extracorporeal Membrane Oxygenation (ECMO) for Initial Management of Post-Myocardial Infarction Ventricular Septal Rupture Associated with Cardiogenic Shock. A Good Long-Term Outcome Starts with a Good Short-Term Result

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Post-myocardial infarction ventricular septal rupture (PIVSR) has previously impacted approximately 1-2% of all myocardial infarctions (MI). Advances in coronary revascularization have reduced this incidence, although mortality remains extremely high.[1] Risk factors for death from PIVSR include female sex, older age, anterior wall MI, arterial hypertension, late arrival to the hospital, first MI, lower left ventricular ejection fraction, cardiogenic shock, elevated white blood cell count, low platelets, liver dysfunction, tachycardia, and lack of surgical repair.[2,3,4] It has traditionally been thought that PIVSR occurs several days after a MI. The clinical presentation of PIVSR is generally correlated with the size of the defect and the presence of other mechanical complications such as free wall or papillary muscle rupture.[1] Shunting of blood from the high-pressure left ventricle to the lower-pressure right ventricle through larger defects results in overflow of blood through the pulmonary vasculature and overall reductions in blood flow out of the left ventricle to the systemic circulation resulting cardiogenic shock. This often occurs even in the setting of a normal ejection fraction.

Data from the SHOCK Trial Registry demonstrated that patients with PIVSR present at a median of 16 hours after MI5. Management of the condition after initial stabilization may include percutaneous transcatheter closure (TCC) or surgical closure or conservative medical management. Conservative management is generally associated with the highest mortality. Mortality may be lower when a surgical intervention can be delayed.[6,7,8] Stabilization followed by delayed definitive management may allow for scar development at the site of infarction, resulting in more durable surgical repair or TCC than operating early when friable tissue complicates management and organ dysfunction may worsen with bypass. Whether surgical or TCC is selected is based upon the availability of devices, anatomy, clinician skill, and patient characteristics.[9]

Mechanical circulatory support (MCS) devices are utilized for the temporary management of patients with cardiogenic shock that is classical, deteriorating, or cardiogenic shock in extremis.[10] These stages of cardiogenic shock may be due to mechanical complications of myocardial infarction, including PIVSR. MCS may bridge patients to clinical definitive therapy. The benefits of temporary MCS can include ventricular unloading, improvement of peripheral perfusion, and decreases in pulmonary capillary wedge pressure.[10] These improvements may facilitate a reduction in pharmacologic agents, which themselves may worsen myocardial damage.[10,11] The American College of Cardiology Solution Set Oversight Committee recommends the use of mechanical cardiovascular support in patients with cardiogenic shock with hypoperfusion and hemodynamic deterioration evidenced by increasing lactate, renal or hepatic dysfunction, and hypotension.[10]

Intra-aortic balloon pumps (IABP) are the most utilized form of MCS, although use varies across institutions.[12,13] The benefit of an IABP in PIVSR lies in a reduction in the isovolumic contraction phase of systole and a lowering of the aortic end-diastolic pressure, facilitating forward flow during ventricular contraction. Blood flow across the VSD is then reduced

although the cardiac output may only increase 0.5-1.0 L/minute.[14] The IABP-SHOCK trial demonstrated no long-term benefit of the use of an IABP for cardiogenic shock complicating myocardial infarction.[15]

The two mechanical modalities that have shown some benefit in the management of advanced cardiogenic shock are the Impella devices (axial flow continuous) and extracorporeal membrane oxygenation (ECMO)10. The Impella CP is placed in a percutaneous manner and can generate flows from 3.0-4.3 L/min while the Impella 5.5 requires surgical implantation can flow up to 6.0 L/minute. The Impella provides direct unloading of the left ventricle, increases in coronary perfusion, and reductions in afterload facilitating improved forward blood flow. Several meta-analysis of the Impella device has shown lower early mortality, less bleeding complications, and lower rates of renal failure.[16,17,18] When the Impella device is compared with ECMO, the Impella provides superior reduction in pulmonary capillary wedge pressure and reductions in left-to-right shunting.[19] Recognizing that there are studies demonstrating improved outcomes and lower rates of complications associated with the use of the Impella device, the question that needs to be addressed is whether the Impella should be the default form of mechanical circulatory support in the setting of PIVSD. We argue that despite the growing body of literature, ECMO remains a superior form of mechanical circulatory support in the initial management of cardiogenic shock refractory to reasonable pharmacologic support.

The existing literature reflects predominantly a retrospective and observational nature for which confounding factors cannot be completely controlled. We do caution though that improved outcomes by does not necessarily translate to adequate or even acceptable outcome and the need for any form of MCS in the setting of PIVSR may indicate that intervention is likely to be futile.

Time and appropriate initial management of cardiogenic shock is the key to survival. The ACC recommends that mechanical circulatory support be guided not only by the degree of cardiogenic shock but by whether the right or left ventricle is the primary cause of shock.[10] In the acute phase, this is not necessarily known and isolated support of the left ventricle, which is the capability of the Impella CP or 5.5, may not provide adequate immediate hemodynamic resuscitation. Veno-arterial ECMO (VA ECMO) provides immediate support of both ventricles as well as the pulmonary system. Immediate complete hemodynamic support eliminates dependence upon the function of the right ventricle to provide adequate filling of the left ventricle that is required for optimal Impella function. Complete cardiopulmonary support eliminates pulmonary edema and hypoxemia as a contributor to additional myocardial strain and injury. Although pharmacologic agents are the primary form of support for patients at risk for or in the early phases of cardiogenic shock, they can increase myocardial oxygen consumption, cause tachycardia and arrhythmias, and contribute to systemic organ dysfunction. ECMO provides the highest level of mechanical circulatory support at up to 7L/minute compared with 6.0 L/min for the highest level of Impella support. Even when an Impella is functioning properly, pharmacologic support of the right ventricle in the form of dobutamine or milrinone may need to continue. Such medications also contribute to systemic vasodilation, requiring the administration of vasopressors, potentially furthering organ damage. A large study of data from the Extracorporeal Life Support Organization (ELSO) database shows that delays in initiation of MCS in patients with cardiogenic shock is associated with worse outcomes.[20]

The increased capabilities of VA ECMO compared with isolated support of the left ventricle provided by the Impella device negates the need to escalate. VA ECMO immediately provides support biventricular and respiratory support. Shock management of the right ventricle with the Impella system requires insertion of an additional Impella or right atrial to pulmonary artery cannula.[21] A recent study showed that Impella use for left ventricular shock was more commonly associated with escalation that use of VA ECMO21. In addition, the Impella 5.5 which can provide higher support (often needed as patients have a mixed shock presentation due to liver congestion from right ventricular dysfunction) requires a surgical cut down and therefore a significant delay before implantation. In comparison VA ECMO can be implemented within minutes and restore end organ perfusion. If a patient is not able to recover from the infarct

preservation of end organ function is critical to preserve candidacy for advanced therapy consideration.

The availability of ECMO as a device of resuscitation continues to grow.[22] ELCO data shows that nearly 600 centers perform ECMO.[23] Safe placement has been demonstrated in the hands of cardiologists,[24] emergency room physicians,[25] and intensivists.[26] This broad spectrum of providers contrasts with placement of Impella devices which are largely limited to cardiologists for percutaneous approaches or surgeons for placement of the Impella 5.5 that requires surgical access to the axillary artery. In the end, it may be that a combination of both devices is ideal; VA ECMO to provide metabolic resuscitation and an Impella device to help unload the ischemic ventricle as reported previously in AMI shock (Unokiet 2024).

It also is critical to remember that no mechanical support device can salvage an unsalvageable situation. As VA ECMO can be used in extremis, including cardiac arrest, it is important for clinicians to be honest with patients, their families, and themselves when choosing appropriate candidates. An infarct VSD represents a very sick patient who has a high likelihood of morbidity and mortality. If a patient is in extremis and there is a possibility of successful recovery or candidacy for advanced therapies (durable LVAD or heart transplant) then VA ECMO is frequently the best support strategy to re-animate a patient.

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Mechanical Support for Bridging Post-Ischemic Ventricular Septal Rupture to Repair: Impella (Abiomed USA) Ventricular Assist Device is Superior to Alternative Mechanical Modalities

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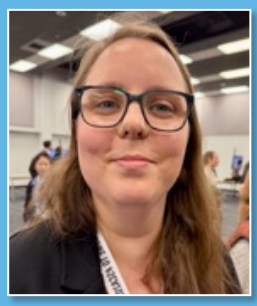

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Ventricular septal rupture (VSR) following acute myocardial infarction (AMI) remains a dreaded and highly morbid complication of acute coronary syndromes, with quoted mortality rates of 41-87%.[1,2,3] The majority of patients present within 48 hours of an infarct, with nearly the rest presenting within 7 days. Presentation includes cardiopulmonary dysfunction and cardiogenic shock.[4,5,6,7] Traditionally, post-infarct VSR (PI-VSR) is considered a medical and surgical emergency, the latter to repair to facilitate management of cardiogenic shock.[3] However, accumulating data support emergent medical management and a delay in surgical or percutaneous repair.[3,4,5,8,9]

Within the first seven days infarcted tissue experiences inflammation, coagulation necrosis, followed by breakdown of the necrotic tissue.[10,11] The dysfunctional myocardium is weakened, experiences higher wall stress and is at risk for expansion and progression.[8,9,10,11,12] Delaying the repair allows time for favorable remodeling and healing of the infarct and peri-infarct area, thereby improving the quality, success, and stability of the repair.[4,5,8,9] Accumulating data report that a delay in definitive repair is associated with repair success, and improved post-operative outcome.[3,5,6,8,9,13,14]

Patients with VSR present with extremes of cardiogenic shock due to left heart failure, and/or right heart failure, the latter of which can be due to a primary myocardial infarct, or secondary to the acute increase in volume and pressure load through a left to right shunt from the VSR, and/or acute increases in pulmonary pressures from left heart failure and/or mitral regurgitation.[4,6,15,16] If cardiogenic shock is not managed emergently and appropriately, the VSR may progress to develop a free wall rupture/pseudoaneurysm, pericardial effusion/tamponade, and multi-organ failure. The most significant mortality predictors across all ages are an inability to repair the VSR defect and cardiogenic shock, the latter being more significant perhaps more so after VSR repair.[4,5,6,17] Successful management of cardiogenic shock before and after repair is associated with a lower mortality.[4,5] A delay in definitive repair allows management of cardiogenic shock and improvement in end organ perfusion/function, an opportunity to obtain more detailed data of the pathoanatomy, and to allow favorable tissue remodeling of the peri-infarct tissues.[3,4,5,8,9]

Reduction of cardiac chamber pressures, distention and stress, while maintaining systemic perfusion are primary goals to manage cardiogenic shock, prevent infarct expansion, and allow favorable tissue remodeling.[6,18,19] Medical management would ideally reduce preload, afterload, and left to right shunt, however, hypotension and cardiogenic shock prompt administration of inotropes and vasopressors which instead increase afterload, contractility, heart rate, chamber pressures, stress, myocardial oxygen consumption, all risking progression and development of greater mechanical complication complexity.[6,18,19] The inclusion of mechanical circulatory support (MCS) into the care algorithm to manage cardiogenic shock has increased over the last 1-2 decades moving beyond intra-aortic balloon counterpulsation (IABP), to include extracorporeal membrane oxygenator (ECMO), and/or transient/temporary percutaneous ventricular assist devices (tVAD). Of the latter, the Tandem Heart (LivaNova Pittsburgh USA) and Impella (Abiomed USA) systems predominate and can be applied for left and/or right heart failure.[20,21,22,23,24,25,26,27,28]

The Impella (Abiomed USA) system is an axial flow ventricular assist device that is placed into the LV with the inflow positioned toward the apex and draws blood from the LV and ejects it just beyond the aortic valve.[32] Its use has improved outcome in patients with cardiogenic

shock.[25,26,27,29,30,31] In cases of PI-VSR, the trend is to delay surgery for at least 7 days perhaps even 21 days. This allows for opportunity to obtain greater detail of the pathoanatomy and permits favorable tissue remodeling are associated with a greater repair success. When considering the hemodynamic effects of different MCS devices, transcutaneous left ventricular assist devices (tLVAD) like the Impella system, more specifically the Impella 5.5, provides more favorable conditions to meet therapeutic goals.[6,18,19,21,22] Compared to the Impella system, both the Tandem Heart and peripheral VA-ECMO, delivers its inflow to the femoral artery and is associated with increased systemic afterload, increased left ventricular chamber pressure, and wall stress potentially impairing tissue remodeling, and possibly risking infarct/defect expansion/progression, and greater shunt flow.[19] These can further overload the right ventricle. The Impella system provides a more favorable hemodynamic solution, and a more optimal MCS device for cardiogenic shock especially when related to a PI-VSR.[6,18,32]

There are a number of Impella devices that provide forward flow from the LV that provide varying hemodynamic support ranging from 2.5L/min to 5.0L/min (Impella CP) for devices placed from the femoral artery, to 5.5L/min for the Impella system surgically placed from the subclavian or axillary artery under fluoroscopic with or without echocardiographic guidance.[32] While the percutaneous Impella CP is only approved for up to 4 days of tLVAD support, the Impella 5.5 is approved by the US Federal Drug Administration provide tVAD support for up to 14 days, and use beyond this is reported.[32] While support up to 14 days is approved, use beyond this is reported.[32] The RP Impella is an axial flow device for the right ventricle that, under fluoroscopic and/or echocardiographic guidance through the right heart with its tip positioned into the main pulmonary artery. If needed, both Impella and RP Impella can be implemented; BiPella (FIGURE 1).

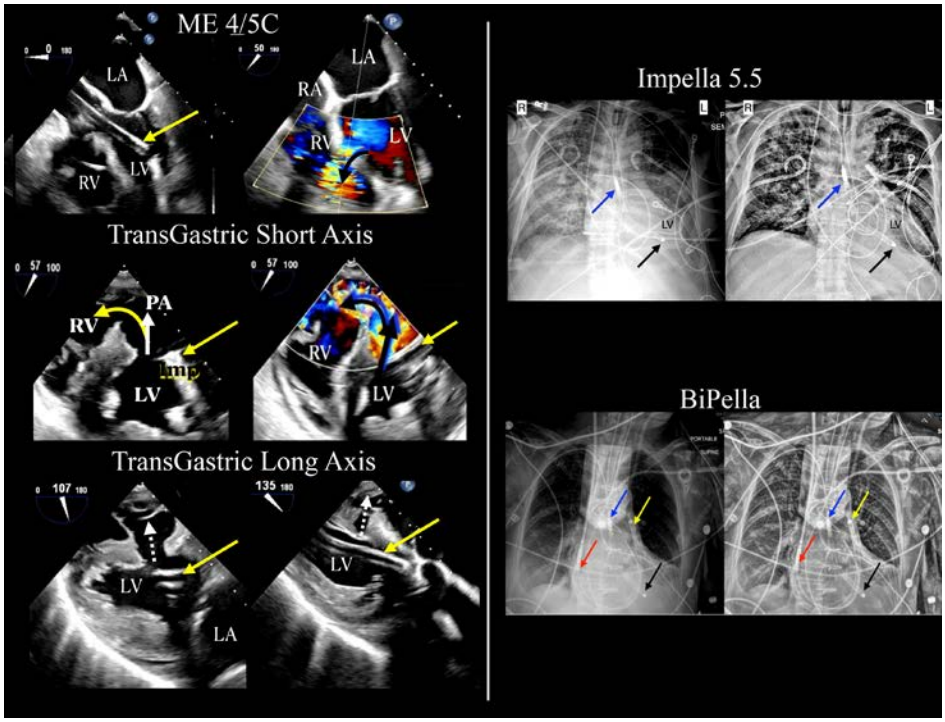


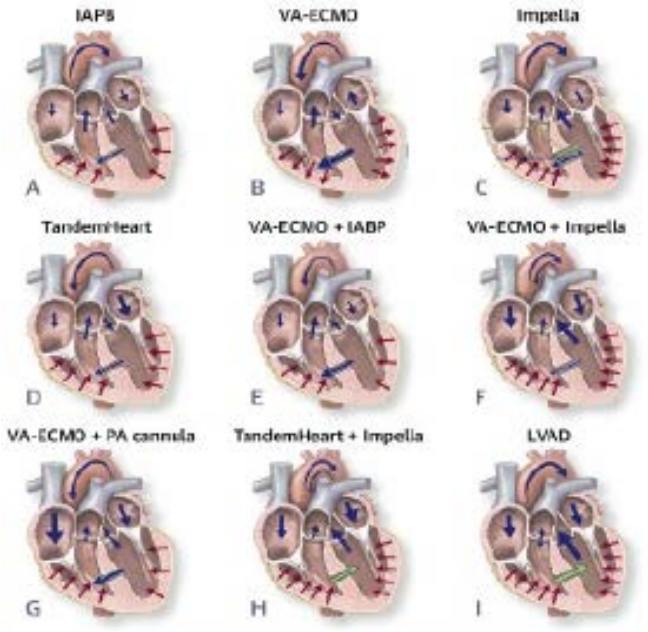
Figure 1: Left of the white line: Mid Esophageal and Transgastric views of the heart highlighting the left and right ventricles and atrium (LV; LA;RV), a pseudoaneurysm (PA), and the impella (yellow arrows). Abnormal VSR and PA flows are seen by the color Doppler, the black arrow in the ME 4/5 view, and the combination arrows in the transgastric 57 degree views. To the right of the white line are chest radiograms with different exposures. In the top pair, the black arrow points toward the tip of the Impella inflow pointed toward the left ventricular apex. The blue arrow points to the outflow just beyond the aortic valve. In the low pair of images, a BiPella is shown. The black arrow points toward the tip of the Impella inflow pointed toward the left ventricular apex. The blue arrow points to the outflow just beyond the aortic valve. The red arrows point to the inflow of the right sided RP Impella flex and the yellow arrows point to the outflow, which is resting in the main pulmonary artery.

CON

For the patient with PI-VSR, the placement of the Impella 5.5 reduces left heart volume, pressure and stress, reduces VSR shunt, has a beneficial impact on RV pressures/volumes, and manages cardiogenic shock.[6,18]

1. Unloads the LV or RV as needed; Impella; RP Impella; BiPella
2. Provides excellent systemic perfusion
3. Provides excellent cerebral blood flow
4. Improves coronary flow
5. Reduces VSR shunt

By comparison to VA-ECMO, for patients with PI-VSR, the Impella 5.5 provides superior reductions in afterload, preload, right and left heart pressures and stress, left and right heart pressures, and reduction in shunt across the VSR, all while providing high cardiac output and systemic blood flow.[6,18] (FIGURE 2). While peripheral VA-ECMO does reduce right heart pressures and provides excellent systemic perfusion/flow, it increases left heart afterload, stress, and shunt.[6,18] The increased left heart strain is not beneficial for peri-infarct remodeling and the increased left to right shunt is detrimental to right heart function and may minimize any reductions in central venous pressures.[6,18]



	Afterload	Preload (PCWP)	PAP	CVP	CO	LV Wall Stress	RV Wall Stress	Shunt
IABP	↓↓	↓	↓	↓	↑	↓	↓	↓
VA-ECMO	↑↑↑	↑↑	↑↑↑	↓	↓	↑↑↑	↑↑↑	↑↑↑
Impella	↓↓↓	↓↓↓	↓↓↓	↓↓	↑↑↑	↓↓↓	↓↓↓	↓↓↓
Tandem Heart	↑↑↑	↓↓↓	↓↓↓	↓↓	↓	↓↓	↓↓↓	↓↓
VA-ECMO/IABP	↑	↓	↑↓	↓↓	↑	↓	↓	↑↓
VA-ECMO/Impella	↓ or ↑↓	↓↓↓	↓↓↓	↓↓↓	↑↑	↓↓↓	↓↓↓	↓↓↓
VA-ECMO/ PA Cannula	↓	↓↓↓	↓↓↓	↓↓↓	↑	↓↓	↓↓↓	↑↓
Tandem Heart/ Impella	↓ or ↑↓	↓↓↓	↓↓↓	↓↓↓	↑↑	↓↓↓	↓↓↓	↓ or Inversion?
LVAD	↓↓↓	↓↓↓	↓↓↓	↓↓	↑↑↑	↓↓↓	↓↓↓	↓ or Inversion?

Figure 2: Comparative hemodynamic between different mechanical support systems and combinations of them. With permission from reference 6: With permission from reference 6: Ronco et al. Mechanical Circulatory Support as a Bridge to Definitive Treatment in Post-Infarction Ventricular Septal Rupture. JACC Cardiovasc Interv. 2021 May 24;14(10):1053-1066. doi: 10.1016/j.jcin.2021.02.046. PMID: 34016403.

CON

Though VSR was an exclusion criterion in the initial approval studies for the Impella 5.5 and remains a contraindication to placement by the manufacturer[33] multiple case reports support the use of Impella in VSR as a bridging modality to successful, delayed surgical repair.[34,35] Although once contraindicated for post-infarct PI-VSR, PI-FWR, and pseudoaneurysm due to concerns of ventricular wall trauma, embolization of friable septal tissue, and shunt reversal,[22] newer data suggest that the Impella can safely provide hemodynamic support, reduce LV pressure and volume, and minimize shunting while facilitating sufficient delay before surgical repair.[25,26,27,36,37,38,39,40] Careful placement using fluoroscopy and/or echocardiography helps to carefully position the Impella device to avoid such complications (FIGURE 1).

Computer simulation comparing MCS modalities in the setting of PI-VSR demonstrated that Impella 5.5 support up to 5 L/min to significantly reduce left to right shunt, increase mean arterial pressure (MAP), reduce pulmonary capillary wedge pressure, and augment cardiac output by up to 42%.[18] While reversal of shunt and consequent hypoxia with the Impella remains a concern, computer simulation up to maximum flow of 5 L/min failed to demonstrate reversal of flow and emerge as the device best capable of reducing left to right shunt when compared to VA-ECMO, IABP, and VA-ECMO+Impella (i.e. ECPELLA).[18]

The Impella MCS increases peripheral and cerebral flow and oxygenation due to increases in systemic perfusion and pressure.[28,41,42,43,44] Increased cardiac systemic blood flow and reductions in LV pressures and volumes help to increase coronary flow.[28,41,42,43,44] By contrast, due to the increased afterload associated with ECMO, coronary blood flow is not increased, potentially risking a mismatch between strain and oxygen demand with oxygen delivery.[28,43,44,45]

Combining the preoperative delay of greater than 7 days, and the continued use of the MCS into the postoperative period, placement of the Impella 5.5 through an upper body artery allows earlier patient mobility.[31] This is not the case with other temporary MCS devices that include femoral cannulae.

All MCS devices are potentially complicated by vascular injury, thromboembolic events, bleeding, end organ dysfunction, peripheral ischemia and stroke.[46,47] While the single cannula Impella systemic might pose lower risk, there are no randomized studies comparing complications. Available data show that both the VA-ECMO and Impella VAD carry similar risks with both being greater than an IABP.[48,49,50] Compared to VA-ECMO, the Impella MCS is less expensive.[51]

VA-ECMO

Without dismissing the value of peripheral VA-ECMO, which provides equivalent systemic flows and is more readily available and can be placed within 45 minutes, it does so at the cost of significant increase in afterload increasing left ventricular end-diastolic pressure (LVEDP), increasing wall pressure, distension, impairing coronary flow, which can negatively impact on tissue recovery, and possibly promote expansion and progression of PI-VSR.[6,18,19,45] In a computer model of MCS in the setting of VSR, left to right shunt was increased to 10 L/min at a maximum VA-ECMO flow of 5.5 L/min.[18] The increased shunt flow would be expected to increase flow and pressure into the RV causing dilation, dysfunction, and congestive multi-organ failure (i.e. congestive hepatopathy or nephropathy).

Interestingly, the increase in afterload to the left heart during VA-ECMO combined with a "pop-off" valve, or reduction of LVOT flow, by the VSR can cause a notable reduction in aortic valve opening, promoting stasis in the aortic root with the formation of aortic root thrombus and increasing the risk of stroke.[45] In addition, the increase left heart pressures may impair coronary flow causing myocardial ischemia.[42,52,53]

Depending on the output of the left heart and the presence of pulmonary dysfunction, a differential hypoxia may be seen during peripheral VA-ECMO that is reflected by lower cerebral oximetry saturations.[42,52,53] This may also be seen when VA-ECMO is added to Impella system.[54,55,56] To offset these issues, additional cannula and configurations of VA-ECMO are entertained, or additional MCS systems are added such as an IABP or an Impella.[28,45,54,55,56,57]

IABP: Simply Not Enough

The use of IABP for patients with cardiogenic shock has not been shown to improve 30 day mortality.[7] Although the IABP provides afterload reduction and improves coronary perfusion, the former is modest at best, at a maximum cardiac output augmentation of only 0.5-1.0 L/min.[18,58,59,60] The IABP is often insufficient to support patients at the extremes of cardiogenic shock or with larger higher flow VSR and significant left to right shunting.[6,18,28] While placement is relatively quick compared to other MCS devices and complications (bleeding, vascular injury, limb ischemia, acute kidney injury) are either less or similar, it lacks equivalent hemodynamic benefits.[48,49,50] Its best use may be when combined with ECMO to offset some of the negative hemodynamic effects of VA-ECMO.[28]

Conclusion

When considering the goals of managing a PI-VSR patient and the MCS technologies, the Impella 5.5 provides hemodynamic advantages to manage cardiogenic shock, reduce myocardial stress, reduce shunt, and stabilize the peri-infarct region while facilitating a desired > 7 day delay in repair. Since MCS devices are continued into the postoperative period with the same goals to stabilize the patient, allow healing of the surgical site, the Impella system continues to have hemodynamic advantage. Given its placement location it may allow earlier mobilization of the patient after surgery.

All considered the decision regarding MCS is patient specific and based on the hemodynamic needs, i.e. left +/- right heart failure, and whether or not a specific device is contraindicated. For biventricular failure clinicians may select to place right and left Impella systems (BiPella) or combine the Impella with ECMO (EcPella).[18,28]

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