SCA NEWS

SOCIETY OF CARDIOVASCULAR ANESTHESIOLOGISTS
Knowledge • Care • Investigation

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February 2025

SOCIETY OF CARDIOVASCULAR ANETHESIOLOGISTS

Kathryn E. Glas
MD, MBA, FASE
President
Society of Cardiovascular Anesthesiologists

PRESIDENT'S MESSAGE

Happy New Year to all! I hope the start of the year has been uneventful for most of you, and your new year's resolutions are still positively influencing your life. My thoughts to all impacted by the fires in LA or the serious snow storms across the Midwest and East coast.

The Board of Directors started the year with a meeting January 10-12 for strategic planning around education and oversight of societal activities. The management team has been busy behind the scenes implementing our plans and ensuring societal activities flow as planned. COVID had a profound impact on in-person meetings, and we learned lessons about virtual and hybrid meetings. Feedback from members indicates the desire to continue with in-person meetings, while also allowing the flexibility of virtual and non-synchronous options. The Online Education Committee is one of our fastest growing, and busiest groups, and we thank this group for SCA University and the great content they oversee. The ARC modules and the question bank were member generated ideas, and they have been well received. Be on the lookout for an update to SCA University that improves your ability to access the content. The Social Media Committee is continuously working to enhance our website and externally facing content, and we look forward to your suggestions on what you need.

The volunteer members of our society are highly engaged in providing us with high quality content in education including lectures, webinars, guideline documents and more. The Research Committee will soon start reviewing submitted grant proposals to continue our support of research in basic, translational and clinical science to advance our specialty. The recent call for volunteers yielded many individuals willing to give time to promote cardiovascular anesthesiology. We have launched an AI Task Force recognizing the key role AI plays in our future. We will soon launch a council for individuals in non-academic/private/community practice. The Board is very interested in engaging our largest member group in shaping the future of the society — look for the call soon!

The Nominating Committee reviewed the applications for Secretary/treasurer, President-elect, Nominating Committee and CME Committee. You should have received your ballot on January 20. Check your SPAM if you don't see it, and please vote!

As always, thank you for placing your trust in me to steward the society for the past 20 months, and as we start the transition towards the next leadership team, I am thankful for this opportunity and look forward to working with all of you in the future to ensure all patients receive high quality care from dedicated cardiac anesthesiologists, many of whom receive ongoing education from our dedicated team.

Sincerely,

Kathryn E. Glas, MD, MBA, FASE

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SCA ECHO 2025

February 20-23

Join Us for SCA Echo, from February 20-23, in the Dynamic City of Atlanta, Georgia!

This four-day conference will immerse you in a series of multidisciplinary panels that explore the critical role of echocardiography in surgical decision-making, especially concerning valvular disease and mechanical circulatory support.

Delve into the complex clinical challenges that present in the operating room and impact surgical strategies. Participate in enriching discussions on transcatheter procedures in structural heart disease, and don't miss our exclusive "Learn from the Experts" sessions.

These sessions will highlight advanced echocardiographic techniques and dissect heart structures to deepen your understanding of echo-anatomic correlations in both transcatheter and surgical interventions.

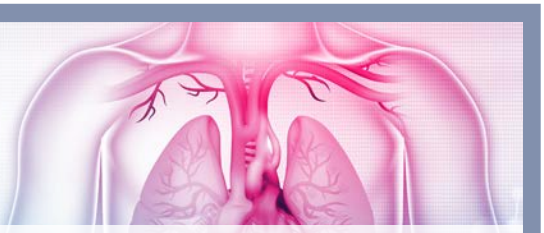
Don't miss this opportunity to enhance your skills and connect with experts in the field of echocardiography!

CLICK HERE **For Registration Details and Program Agenda**

Co-Chair
Alina Nicoara, MD, FASE

Co-Chair
Charles Nyman, MBBCh

Vice-Chair
Kimberly Howard-Quijano, MD



TAS2025

THORACIC ANESTHESIA
SYMPOSIUM & WORKSHOPS

APRIL 25 | Montréal, Canada

Please Plan to Join Us at the 13th Annual Thoracic Anesthesia Symposium and Workshops in *Belle* Montreal, Canada!

We are excited to see you there for a robust exchange of ideas, techniques, and advances in the field of Thoracic Anesthesia.

We recognize how valuable your time is and the wide array of choices available to you for continuing medical education. In addition to didactic sessions presented by international experts in Thoracic Anesthesia, the Thoracic Anesthesia Symposium Planning Committee is also excited to offer a selection of updated and new workshops featuring hands on experience with 3D printed models, precision ultrasound guidance, and augmented reality technology.

As always, we aim for the Symposium to provide you with critical updates on relevant topics in Thoracic Anesthesia, to enhance your learning of important techniques and skills, to promote discussion and debate of controversial topics in our field, and to facilitate networking. We are also pleased to offer novel research and challenging case presentations in both poster and oral presentation formats as well as several problem-based learning discussions. Each session is thoughtfully designed to present clear and timely information pertinent to our unique subspecialty.

During the next months leading up to our meeting in Montreal, please check out the SCA DocMatter Thoracic Channel (DLT Exchange) for lively discussions curated by our Planning Committee members along with teasers of our 2025 Symposium content.

We look forward to reconnecting familiar colleagues to meeting new ones!

On behalf of the SCA and the 13th Annual Thoracic Anesthesia Symposium and Workshops, we are so excited see you in Montreal!

CLICK HERE **For Registration Details and Program Agenda**

Sincerely,
Rebecca Klinger, MD MS
Chair, Thoracic Anesthesia Symposium and Workshops Program Committee

Join Us April 26-29, 2025 for the Society of Cardiovascular Anesthesiologists 47th Annual Meeting and Workshops in Beautiful Montreal, Canada

Don't miss a top-notch educational program and opportunities to congregate with friends and colleagues while surrounded by a unique mix of European and North American culture.

Enjoy small group sessions, workshops, and presentations and panel discussions on all the hot topics and developments in our field. The program will address the latest research, as well as clinical controversies in our practice, with experts from the world of cardiology, cardiothoracic surgery, perfusion, critical care medicine, regional anesthesiology, law, and finance enriching our panel discussions. The workshops are hands-on opportunities to learn cutting edge technology from masters in the field in smaller, more interactive settings. Problem-based learning discussion sessions offer opportunities to learn by working through clinically challenging cases in a focused, small group setting led by experts.

Research focused sessions will have experts presenting and discussing latest trials and cutting-edge work, the work of our SCA members will be presented in the "Best of Meeting" sessions and in abstract presentations. A brand-new Cardiovascular Outcomes Research in Perioperative Medicine (COR-PM) Research track will be included in the Annual Meeting and offer high level content for the serious researchers among us. Echocardiography CME hours will be offered in general sessions and workshops, and the true echo-geeks among us won't want to miss the discussion at the SuperEcho session.

Finally, we have a specific program designed for trainees, with unique sessions geared toward the educational and professional development needs of our medical students, residents, and fellows.

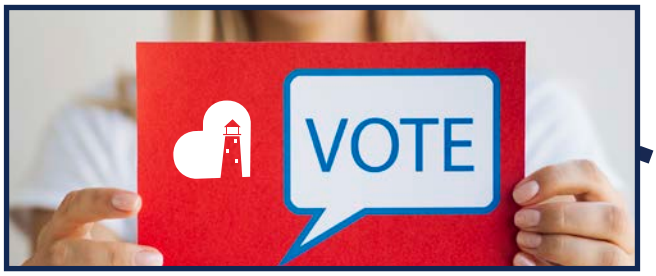
Full Program details will be forthcoming but save the date and plan on joining us in Montreal as we come together to share our knowledge and experience in our chosen specialty.

CLICK HERE **For Registration Details and Program Agenda**

See you there!

Jonathan Ho, MD FASE, Chair, Scientific Program Committee 2025
Stephanie Ibekwe, MD, MBA, MPH, MS, Vice Chair, Scientific Program Committee 2025

SCA 2025 Elections
VOTING IS NOW OPEN!



The 2025 online elections for SCA leadership positions are open through March 11. The candidates are running for the following positions: Active and Associate members in good standing will receive a personalized link to their primary and secondary email address to cast their vote.

President Elect
Secretary / Treasurer
Director-at-Large
Nominating Committee
Continuing Medical Education (CME) Committee Member

The SCA Nominating Committee, chaired by Immediate Past President Dr. Andrew D. Shaw, is pleased to endorse the following candidates for the 2025 election cycle:

President-Elect

Douglas Shook, MD, FASE
Brigham and Women's Hospital

I am running for the position of President-Elect of the Society of Cardiovascular Anesthesiologists (SCA). As the current Secretary/Treasurer and having served two 3-year terms on the Board of Directors, I have developed a strong understanding of our society's mission and its members' needs. My leadership experience also includes the roles of Co-Director of SCA Echo, Chair of the Fellowship Program Directors Council, and co-developer of the Kaplan Leadership Development Award, designed to cultivate future leaders in our profession.

Through these positions, I have demonstrated my commitment to member education, the advancement of future cardiovascular and thoracic anesthesiologists, and leadership development within our field.

Secretary/Treasurer

James (Jake) Abernathy III, MD
John Hopkins University School of Medicine

Dr. Abernathy is a Professor, Division Chief of Cardiac Anesthesiology, and Vice Chair for Strategy at Johns Hopkins University. Dr Abernathy has served the SCA for nearly 20 years, including the 2 terms on the Board of Directors, Fellowship Program Directors (Chair), Annual Meeting Program, STS Database, Kaplan Leadership (co-Chair), Board Certification Task Force, and FOCUS (Chair). He was the inaugural Chair of the Quality and Safety Steering Committee. Dr Abernathy has published over 90 peer reviewed publications and delivered nearly 100 national and international talks. Funded by AHRQ, he is collaborating with human factors engineers to redesign healthcare and improve safety. After completing medical school at the University of Alabama at Birmingham he did residency and fellowship at the Brigham and Women's Hospital in Boston, MA. Prior to joining Hopkins, Dr. Abernathy was Division Chief at the Medical University of South Carolina.

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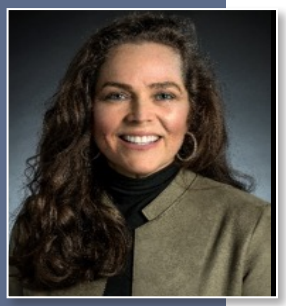


Mary Beth Brady, MD

Johns Hopkins University School of Medicine

I still cannot believe this but my first SCA participation was at the 1999 Annual Meeting. Since, I have been lucky enough to participate in the SCA via a multitude of meetings, committees, taskforces, councils, and the Board of Directors. This culminated in my role as chair of the 2024 Annual Meeting which, for the first time, was in collaboration with our surgical colleagues! My goal is not to provide a laundry list of my past SCA experiences. Rather, I prefer to mention that throughout these roles, my battle-cries have always been, "What is best for our members?" and "How can members engage?" I am not a proud person but love that I cannot begin to count the number of colleagues I have encouraged, sponsored, mentored to get involved in the SCA. Suffice it to say, I believe in leaders that serve others. I humbly strive to do so.

Michael P. Eaton, MD, FASA

University of Rochester Medical Center

Dr. Eaton served as Chair of Anesthesiology and Perioperative Medicine at the University of Rochester in 2024 for 13 years, stepping down September 30, 2024. Dr. Eaton completed his residency in Vermont and a cardiac fellowship at the University of Michigan. He has been a cardiovascular anesthesiologist in Rochester for 29 years, recently focusing on pediatric cardiac anesthesia. He has authored over 40 peer-reviewed publications with primary interest in the hematologic effects of cardiopulmonary bypass. Mike has served the SCA in many capacities since joining in 1994, including the SCA Electronic Communications Committee (ECC) (Chair 2007-2011), the Task Force on Educational Activities, and the Executive and Advisory Boards of the Women in Cardiothoracic Anesthesia SIG. He served on the Scientific Program Committee from 2011-2020, chairing the committee for the 2019 and 2020 meetings. Dr. Eaton was a member of the SCA Board of Directors from 2011-14, 2016-2020 and 2021-2022.

Director-at-Large

Theodore J. Cios, MD, MPH, FASA, FASE

Penn State College of Medicine

Dr. Cios is an adult cardiothoracic anesthesiologist at Penn State Health Hershey Medical Center. He obtained his medical degree from the Ohio State University after which he completed residency and fellowship training at Penn State. As an Associate Professor, he serves as the Program Director of the Adult Cardiothoracic Fellowship and is heavily involved with teaching and has several peer-reviewed publications on topics pertaining to cardiothoracic anesthesia and echocardiography. Dr. Cios has sat on several committees at Penn State including the University Faculty Senate and Chaired an Institutional Review Board. He has been on SCA committees since 2017 as a member of the Clinical Practice Improvement Project Sub-Committee and will continue to serve on the Guidelines and Standards Sub-Committee into 2023. He has given several national and international lectures and serves as a manuscript reviewer for several journals.

Charles Nyman, MBBCh

Brigham & Women's Hospital

As a member of the SCA Board of Directors, I would strive to meet the needs of our membership, the society's mission, and to advocate for our role in the care of cardiovascular patients. Since 2010, I have been a proud member and servant leader of the SCA. I am currently the course Co-Director for SCA Echo Week, and served terms on the Scientific Program Planning Committee, the Echo Week program committee, and the Guidelines and Standards Committee. This foundation of experience and voluntary commitment to the SCA has allowed me to continue my passion to educate our membership, train future cardiothoracic anesthesiologists, support research and serve our profession.

Daryl Oakes, MD

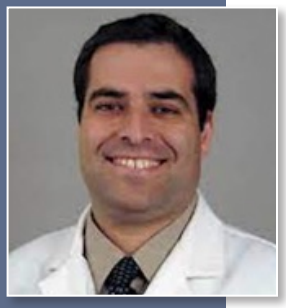
Stanford University

Dr. Oakes is a Clinical Professor at Stanford School of Medicine with over 15 years of clinical experience in cardiothoracic anesthesiology. She is program director for the Stanford adult cardiothoracic anesthesiology (ACTA) fellowship and directs the Stanford Anesthesiology Perioperative Echocardiography Services. Dr. Oakes is also Associate Dean of Post Graduate Medical Education and the Stanford Center of CME. The focus of her academic work has been the education and training of anesthesiologists at all levels of practice. She lectures nationally on a range of topics related to transesophageal echocardiography, cardiothoracic anesthesiology, and education. Dr. Oakes is a passionate mentor to both trainees and colleagues and has created multiple programs to support physician professional development. She co-founded and chairs the SCA Women in Cardiothoracic Anesthesiology (WICTA) Special Interest Group and was recently recognized for her work supporting women medical professionals with the 2021 Women in Medicine (WIM) #SheForShe Award.

Alessia Pedoto, MD

Memorial Sloan-Kettering Cancer Center

Dr Pedoto is a fellowship-trained, thoracic anesthesiologist working at Memorial Sloan Kettering Cancer Center in New York City. She received her medical degree In Italy, from the University of Milan, retrained in anesthesia at New York-Presbyterian Weill Cornell, and did a thoracic anesthesia fellowship at Brigham and Women Hospital. In addition to her clinical work, she is a teacher and a researcher. She has been a member of the SCA for the past 20 years and involved in the Thoracic Anesthesia Symposium for the Society of Cardiovascular Anesthesia since 2013, first as a member of the planning committee, followed by the Abstract and PBLD Coordinator- Scientific Vice Chair and Chair. Dr Pedoto is active within several local and national organizations. She is a member of the CME Committee for the New York State Society of Anesthesia, and the ASA Educational Track Subcommittee in cardiac anesthesia. She was the TAS liaison for the Online Education Subcommittee and the Abstract Committee of the SCA.



Jacob Raphael, MD, FAHA

Thomas Jefferson University Hospital

Dr. Jacob Raphael has been an SCA member since 2003 and currently chairs the SCA Patient Blood Management Sub-Committee. During his longstanding service, Dr. Raphael has been honored to work with society members in advancing SCA's mission of promoting excellence in cardiovascular medicine. He is a regular participant both as faculty and attendee at the SCA's Annual Meeting, Echo Week, and the International Congress of Cardiothoracic and Vascular Anesthesia. He served on the SCA's Scientific Program Planning Committee, Research Committee, SCA/STS Database Sub-Committee and Continuous Practice Improvement Committee. He is committed to promoting diversity and inclusion within SCA and is a strong proponent of interdisciplinary collaborations to advance the care of cardiovascular and thoracic surgical patients. Additionally, he also serves on the leadership committee of the American Heart Association Collaborative of Cardiovascular Surgery and Anesthesia. Dr. Raphael would be honored and humbled to serve as Director at-Large on the Board of Directors.

Jochen Steppan, MD, DESA, FAHA, FASA

Johns Hopkins University School of Medicine

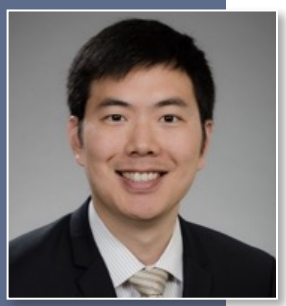
Dr. Steppan is an Associate Professor at Johns Hopkins University, performing both adult and pediatric cardiac anesthesia. He serves as the Director for Perioperative Medicine, High Risk Cardiovascular Disease. After completing medical school at the University of Heidelberg in Germany he did his residency and fellowships at the Johns Hopkins University. Dr Steppan has served the SCA for 15 years. He is a founding member of the Cardiovascular Outcomes Research in Perioperative Medicine (COR-PM) Conference at the SCA meeting to advance clinical outcomes research in cardiovascular medicine, by focusing on mentorship for junior faculty, and using an inclusive and diverse approach. He has served on the Research Committee, the International Council, and has created multiple sessions for the annual meeting. Dr Steppan has published over 80 peer reviewed publications and delivered over 60 national and international talks. Funded by the NIH, he is studying the molecular mechanisms underlying pulmonary hypertension.

Nominating Committee

Choy Lewis, MD

Northwestern University

Dr. Lewis is an Associate Professor of Anesthesiology and Chief of the Division of Cardiac Anesthesiology at Northwestern Memorial Hospital, Feinberg School of Medicine of Northwestern University. She previously served as the Program Director for the Adult Cardiothoracic Anesthesiology (ACTA) Fellowship at Northwestern University as well the Medical Director for cardiac, thoracic, vascular, and transplant surgery at Northwestern Memorial Hospital. Dr. Lewis completed her residency in anesthesiology at Brigham and Women's Hospital and ACTA fellowship training at Weill Cornell Medical Center. She has been a member of the SCA her entire professional career and has served the Society in several capacities while also benefiting from everything that the Society offers. Dr. Lewis is currently a member of the SCA's Member Engagement Committee and the Scientific Program Committee. She is also a member of the Simulation Special Interest Group and WICTA Special Interest Group, where she previously served as the Member Engagement Liaison and is now an Executive Committee Member-at-large.



Richard Sheu, MD

University of Washington

Dr. Sheu is a board-certified cardiothoracic anesthesiologist and the Director of Perioperative Echocardiography at the University of Washington Medical Center. He is also an Associate Professor of Anesthesiology and Pain Medicine and the Program Director of the Adult Cardiothoracic Anesthesiology Fellowship at the University of Washington School of Medicine. Aside from delivering safe and effective anesthetics to patients with cardiovascular and pulmonary diseases during complex open-heart surgeries, Dr. Sheu is an expert in utilizing advanced transesophageal echocardiography techniques in the perioperative period for both diagnostic and monitoring purposes. As treatment options rapidly evolve, he has established himself as a key member of the Structural Heart Team at the University of Washington Heart Institute by providing high-quality imaging guidance and interpretation for transcatheter therapies. Dr. Sheu received his medical degree from New York Medical College and completed his anesthesiology residency with academic distinction at Tufts Medical center in Boston. He further specialized in adult cardiothoracic anesthesiology at the Cleveland Clinic.

Continuing Medical Education (CME) Committee Member

Dalia Banks, MD, FASE

University of California San Diego

Dr. Dalia Banks Professor at the University of California San Diego has been an active member of the SCA since 1998. She finished her anesthesia training at Yale-New Haven Hospital, Cardiac Anesthesia Fellowship at Beth Israel Deaconess in Boston. In October 2005, she joined UCSD. She served as the UCSD Cardiothoracic anesthesia fellowship director for 11 years, division chief of cardiothoracic anesthesiology for 9 years. She is currently serving as the Vice-Chair of Cardiovascular Anesthesia Academic Affairs. She is on the editorial board of the Journal of Cardiovascular Anesthesia where she is a section editor. Additionally, she is a member of the ASA Committee on Cardiovascular Anesthesiology and the ASA Educational Track Sub-Committee on Cardiac Anesthesia. With respect to the SCA, she served as the chair of the Newsletter subcommittee for 4 years. She currently serves on the SPC committee. Dr. Banks is committed to education and has established several CME accredited education courses at UCSD, and she is chaired the California Society of Anesthesiologist Annual Meeting.

ELECTIONS

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]

3SCTS 2025 — Tri-Society Cardiac and Thoracic Symposium & IACA

For symposium details, please visit https://3scts2025.com

2026 SF Match

In-order to provide more consistency and predictability to the ACTA fellowship application process, the ACTA programs participate in a common application and match process provided by SF Match for recruitment.

Please note only program directors may begin the exception agreement process and may do so by clicking **HERE.**

DEI CORNER

Turning Aspirations into Reality: A Scholar's Story

Rachel Reindorf, MD, MPH1; Seema P. Deshpande, MBBS, FASA.[1]
[1] Department of Anesthesiology, University of Maryland School of Medicine, Baltimore, MD

Demographics of the United States (US) are everchanging. However, the population is changing much more rapidly than the demographics of US physicians, leading to physician-patient disconcordance. Although there have been many efforts to get underrepresented minorities (URMs) into healthcare, URMs still remain a small percentage of US physicians. Time and time again, data shows that patients receive better care when treated by physicians who understand their cultural norms, languages, and traditions.[1,2]

The Society of Cardiothoracic Anesthesiology (SCA) Diversity, Equity, and Inclusion (DEI) Junior Resident Scholar Program was created by the SCA's DEI Committee to pave the way for URMs to get early exposure and experience in cardiac anesthesiology. This program gives ten URM residents in their first clinical anesthesiology (CA-1) year a $1000 grant and the opportunity to attend the SCA annual meeting, allowing the residents a chance to present a poster or abstract and network with current residents, cardiothoracic (CT) anesthesiology fellows, and CT anesthesiologists. The early exposure to CT anesthesiology provides more insight into a career as a CT anesthesiologist and the fellowship application process. The article by Dr Mary Arthur- "The SCA Junior Resident Scholar Grant: Addressing the Pipeline Issue," published in the May 2023 SCA newsletter- discusses the background of the SCA Junior Resident Scholar Grant creation.[3] The SCA has thus far recruited two incredible cohorts of residents and is ready to take on their third cohort.

About 72% of the 2023 cohort had their cardiothoracic anesthesiology rotation after attending the SCA Annual meeting (Figure 1), while 86% planned to apply for fellowship (Figure 2). All 2023 DEI resident scholars surveyed reported that the grant played a pivotal role in their decision to apply for a fellowship (Figure 3), a testament to the effect of mentorship, early exposure, and opportunities for networking in fostering interest in different specialties and shaping the careers of resident physicians.

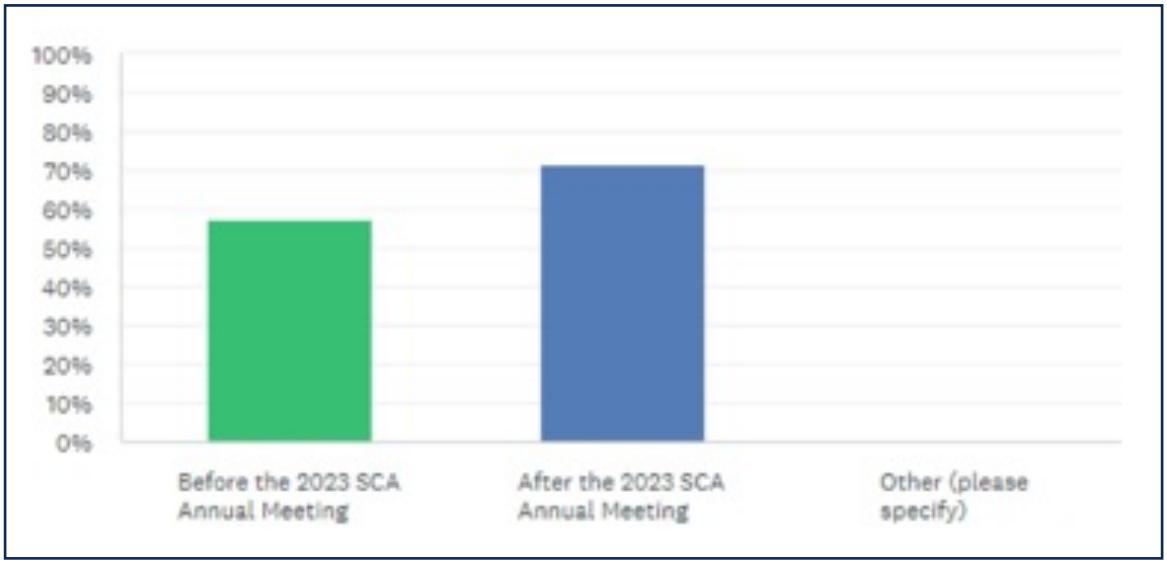


Figure 1. Cardiothoracic Anesthesia rotation before vs. after SCA annual meeting

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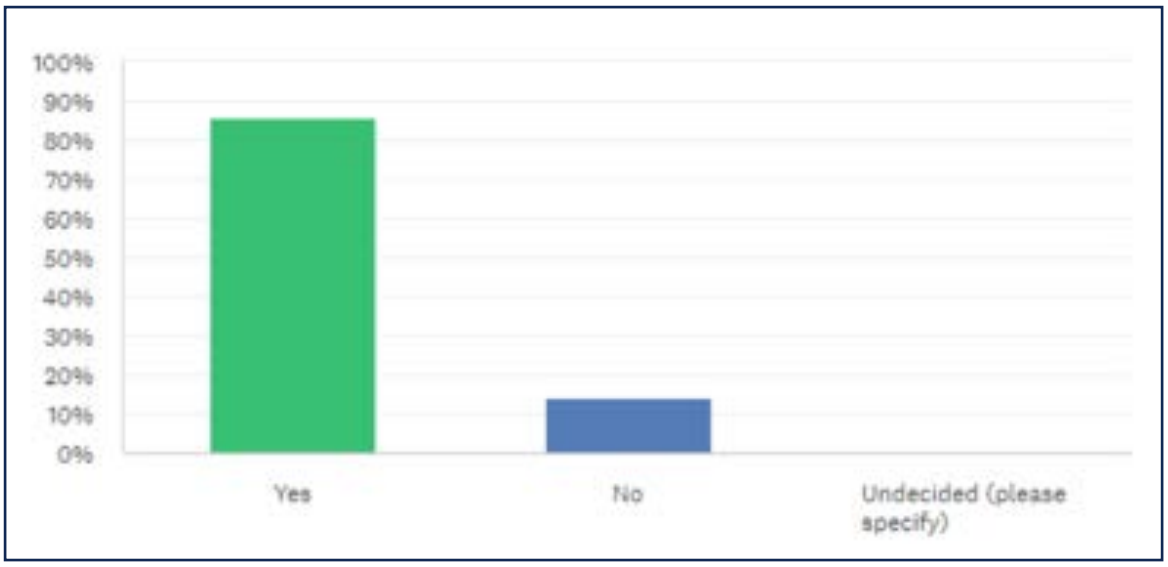

Figure 2. Percentage of 2023 SCA DEI Junior Resident Scholars planning on applying to fellowship

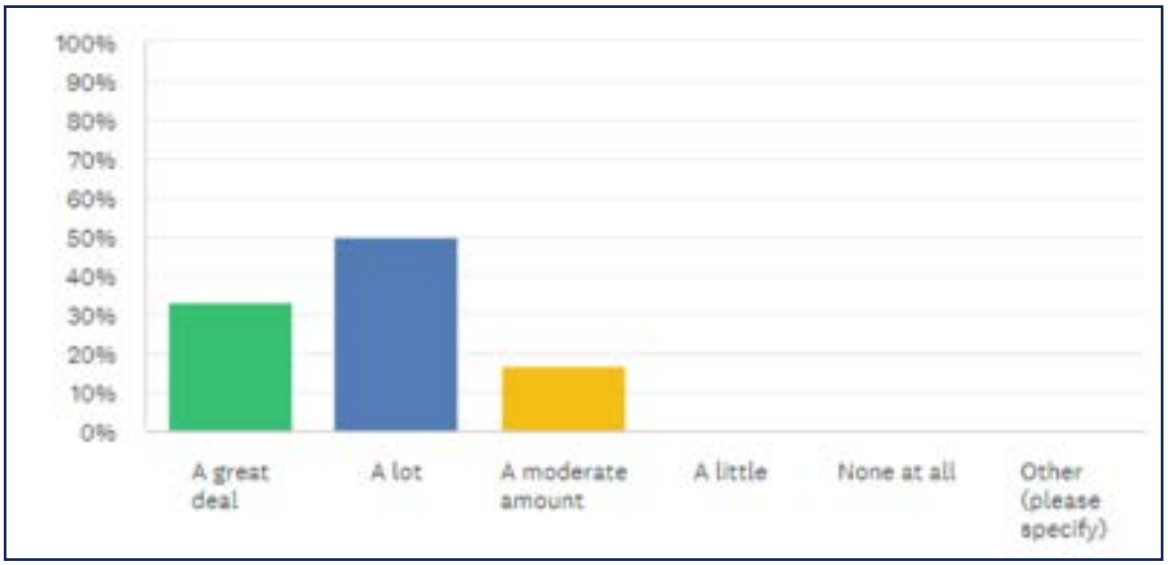

Figure 3. Impact of SCA DEI Junior Resident Scholars Grant on scholars' decision to apply for a fellowship

Perspective from One of the Scholars

My name is Rachel Reindorf. I was part of the first SCA DEI Junior Resident Scholars cohort in 2022. I had come into residency thinking I was going into chronic pain. I did not have much experience in cardiothoracic anesthesia, so I initially had not considered the field as an option for me. I was fortunate to have early exposure to cardiothoracic anesthesiology during my residency at the University of Maryland Medical Center in Baltimore, Maryland. I found that I enjoyed the fast-paced environment of cardiothoracic anesthesiology much more than I had anticipated. One of my attendings sent me an email encouraging me to apply for the SCA DEI Junior Resident Scholar's Grant. I was fortunate to receive the grant and had the opportunity to attend the SCA annual meeting as a CA-1resident. In addition to the opportunity to network at the annual meeting, the program also connected me with a mentor. My mentor guided me through poster submissions and the fellowship application process. Now, as a CA-3 and incoming 2025-2026 Johns Hopkins Adult Cardiothoracic Anesthesiology Fellow, I look back on

the program fondly, as it has positively influenced my career trajectory.

My experience with SCA DEI Scholars has highlighted the significance of mentorship and diversity in medicine. It enables trainees to cultivate relationships with physicians in fields they once might not have envisioned themselves in. Having the chance to ask questions and engage in discussions about challenging cases, projects, or career advice serves as a constant reminder that you've been embraced by a community. I am continually reminded that at any stage of training, one can be both a mentor and a mentee. I aspire to keep working with DEI initiatives and serve as a pillar of this community.

References

1. Grumbach, K., & Mendoza, R. (2008). Disparities In Human Resources: Addressing the Lack of Diversity in the Health Professions. Health Affairs, 27(2), 413–422. https://doi.org/10.1377/hlthaff.27.2.413
2. Guilbert, J.-J. (2006). The World Health Report 2006: working together for health. Education for Health (Abingdon, England), 19(3), 385–387. https://doi.org/10.1080/13576280600937911
3. Arthur, M. " The SCA Junior Resident Scholar Grant: Addressing the Pipeline Issue" on behalf of the Society of Cardiovascular Anesthesiologists' DEI Committee; SCA Newsletter Volume 36, Number 20 May 2023.

SPOTLIGHT

Awesome Woman Interview

Stephanie Ibekwe, MD, MBA, MPH, MS

Assistant Professor of Anesthesiolog
Baylor College of Medicine
Houston, Texas

Dr. Stephanie Ibekwe is an Assistant Professor in the Department of Anesthesiology, Cardiothoracic Division, at Baylor College of Medicine in Houston, Texas. At her primary site, Ben Taub Hospital, Houston's largest safety-net hospital, Dr. Ibekwe serves the Cardiovascular, Thoracic, advanced Oncologic, and Trauma anesthetic needs of the medically underserved residents of Houston, Texas. She is Ben Taub's Director of Quality Improvement and Service Chief of Cardiovascular Anesthesiology. She serves under the esteemed leadership of her Chairman, Dr. James Anton, and Chief of the Department, Dr. Lisa Wofford.

Dr. Ibekwe holds several national leadership roles, including committee member on the American Society of Anesthesiologists (ASA) Quality Management and Departmental Administration Committee, board member and current Vice Chair of the Scientific Program Committee in the Society of Cardiovascular Anesthesiologists (SCA), and a member of the editorial board of the Journal of Cardiothoracic and Vascular Anesthesia. She collaborates with clinical leaders worldwide and leverages her public health, business administration, and transformational leadership expertise to develop and implement sustainable perioperative quality improvement initiatives.

Dr. Ibekwe is an educator, mentor, speaker, clinical leader, and author. She leverages her educational background in public health and business administration, expertise in healthcare organizational leadership, and advanced certification in transformational leadership to build sustainable perioperative quality improvement processes nationally and internationally. Her published works are in ***Perioperative Medicine, Current Opinion in Anesthesiology, Anesthesiology, Anesthesia and Analgesia, the ASA Monitor, and the Journal of Thoracic and Cardiovascular Anesthesiology.***

1. What led you to become a Cardiovascular/Thoracic Anesthesiologist?
Anesthesiologists have the privilege of studying, interacting with, and changing human physiology in real-time. I think of anesthesiologists as engineers of human physiology who must adapt to the dynamic changes of the human body as it responds to the stress of surgery.

As a resident in training, these aspects of anesthesiology were demonstrated in the cardiovascular operating room. I remember the first time I was in the operating room when the patient was placed in deep hypothermic cardiac arrest. It felt like the room was completely silent when the perfusionist turned off the cardiopulmonary bypass machine. I watched the surgeon operate, and then at the end of the repair, the OR came back to life as the patient's blood began to circulate until finally, the anesthesiologist and surgeon worked in concert with the perfusionist to wean the patient from cardiopulmonary bypass. It was like watching a beautifully choreographed dance; I wanted to learn more about the field after that.

2. How did you hear about the SCA?
As a resident at Emory University, I heard about the SCA from a few of my amazing attendings, Drs. Kathy Glas, Michele Sumler, Matthew Klopman, and Abimbola Faloye. They encouraged me to send case reports from my difficult cases to be considered for acceptance for presentation.

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As a cardiothoracic anesthesiology fellow at Johns Hopkins, I became more involved with guidance from Dr. Mary Beth Brady. She encouraged all the fellows to submit their cases to the fellow program and worked with us to fine-tune our presentations. She, along with several of my other attendings, namely Drs. Megan Kostibas, Michael Grant, Jochen Steppan, and many others, encouraged me to be an active member of the SCA.

3. What roles have you held for the society?
I have had the opportunity to serve in many capacities in the SCA. I served as a member of the Enhanced Recovery after Cardiovascular Surgery Task Force, helped review abstracts and problem-based learning discussion submissions, served on the scientific program committee as a member, and subsequently coordinated the SCA workshops and problem-based learning discussions. I am currently the Vice Chair of the Scientific Program Committee and the Chair of the Abstract Review Subcommittee.

I was elected as an early-career Board of Directors member and served for two years. I currently sit on the Board of Directors due to my leadership role on the Scientific Program Committee. I am also an executive board member/member-at-large of the Women in Cardiothoracic Anesthesiologists special interest group of the SCA.

4. What is one of your greatest achievements as a Cardiovascular/Thoracic Anesthesiologist?
I'm not sure what my greatest achievement is, honestly. I'd say that I feel like I've achieved something good when I can help one of my patients feel calmer about their upcoming surgery or help them achieve an "aha" moment about their disease process and how we will approach it in the operating room.

My favorite thing to do is empower my patients in the moments right before they entrust me with their lives. I enjoy introducing the concept of enhancing surgical recovery before they enter the OR. I love to see them begin to hope for a good outcome and feel a sense of agency over their health when I tell them that we can start the recovery process long before they arrive at the hospital.

5. Do you have any advice for fellows and residents?
Yes. Your journey in medicine is a marathon, not a sprint. It's okay if you don't know exactly what you want to do for the rest of your career. You may be in a position where you know that you love cardiovascular anesthesiology, but you aren't sure what practice type you want to pursue, and that's perfectly normal. Tackle the steps that it'll take to land a fellowship. Arrive early, be a team player, work hard, and have a positive attitude, and you'll be successful. In your interactions with patients, take a moment to learn something about them and imagine what it might be like to trade places with that person. You'll be surprised how this small act changes how you think about your patient as you care for them. Our patients trust us to care for them; we shouldn't take that trust lightly.

6. Have you experienced any difficulties as a woman in the field?
Yes, of course. I've attempted to learn something from the difficult parts of being a woman in the field. Sometimes, a situation requires that I change my perspective, my behavior, or my expectations. Some difficulties are easier to navigate than others. I do my best not to navigate things in isolation. When dealing with a problem, I usually confide in a trusted colleague, my husband, or my friends to gain insight and ideas on obtaining a solution or resolution.

7. Do you have any advice for other women in the field?
Being a woman in cardiothoracic anesthesiology is a superpower. Women tend to be multitaskers, collaborators, and detail-oriented, which are some of the most important characteristics of great cardiovascular and thoracic anesthesiologists. Being a leader in cardiovascular anesthesiology looks different for me than it would for a man, and I tend to lean into those differences.

I'm thankful to the women who came before me in the field, who paved the way for my generation, and those who will come after me to lead in our field. I've been blessed to have women who have served as mentors, sponsors, and advocates, and I've needed their support in different ways throughout my career. Many of the women who have mentored and coached me are members of the SCA. The special interest group, WICTA, is a great place to find colleagues and mentors to walk alongside you on your journey in cardiothoracic anesthesiology.

8. How do you balance work and personal life?
I think that work/life balance is a misnomer. I am a wife, mother, friend, sister, physician, author, speaker, mentor, etc. I cannot function at 100% in all those roles simultaneously, and I've decided that's okay. When I'm in the operating room fighting to save my patient's life, I am 100% a cardiovascular anesthesiologist. I use my training and experience to provide my patients with the best care I can deliver. When I'm at home with my family, I give them 100% of me, whether having a bake-off with my kids' friends in our neighborhood or planning a romantic dinner with my wonderful husband.

9. What is something you enjoy doing outside of work?
When it comes to taking care of myself, I approach it by taking care of my mind, soul, and body.

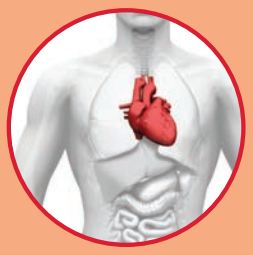
Mind— I enjoy being creative. I love to write/journal and read self-improvement books. I've been learning to paint and play the guitar for years and consider myself a cheerful novice at both.

Soul— I am a Christian and exercise the spiritual disciplines of praying and studying the Bible daily (or nearly so). This helps me shift my focus from myself to what I believe God has called me to do with my life.

Body— I challenge myself to run 5Ks, 10Ks, and, for the second year in a row, a half marathon annually. I'm challenged to stay consistent with my training by training for various races throughout the year.

10. Would you change anything about the path you took to get to where you are now?
No, I wouldn't change anything. Every failure and success have brought me to this point.

11. What was the best piece of advice you received?
Dr. Sandeep Markan, my chief in my first position out of fellowship, now Chief of Staff at Ben Taub Hospital, gave me great advice. I asked him what I could do to ensure my success as a cardiac anesthesiologist as a brand-new attending, and he said, "F/8 and Be There." He told me that one of the most successful photographers, Arthur Fellig, took world-renowned photographs. When asked how he managed to take these amazing pictures, he stated that he used the best default settings of his camera and made sure to be present to take photos. Dr. Markan's words have helped me along the way in my career. While perfectionism can be paralyzing, being prepared tends to be a motivator for me. I stay prepared, and I show up to do the work. Doing this has worked well so far.



ECHO CME

LEARNER NOTIFICATION

The SCA Newsletter is pleased to provide CME for perioperative transesophageal echocardiography. ECHO CME will be available to SCA members in each issue.

Society of Cardiovascular Anesthesiologists
Activity Title: 2025 SCA Echo Corner (TEE Evaluation of MV Repair)
Release Date: 2/3/2025
Expiration Date: 2/3/2027
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.

- Cardiothoracic anesthesiologists may have limited expertise in precise quantification of regurgitation severity (e.g., effective regurgitant orifice area, vena contracta width)
- Cardiothoracic anesthesiologists can be inexperienced in diagnosing systolic anterior motion (SAM) or predicting its likelihood based on pre- and intra-repair findings.

Needs that Underlie the Gap
There is a need to provide education to clinicians on how to perform echocardiographic assessment of mitral valve pathology, including 3D qualitative and quantitative of the MV. There is a need to provide clinicians with relevant echocardiographic techniques to determines the risk of SAM prior to MV repair and identify SAM post MV repair.

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ECHO CME

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.

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

- Evaluate the anatomy and function of the mitral valve, including leaflets, annulus, chordae tendineae, and papillary muscles.
- Assess risk of complications such as systolic anterior motion (SAM) of the mitral valve or left ventricular outflow tract obstruction.
- Confirm the adequacy of the repair, including residual mitral regurgitation, and identify complications such as mitral stenosis or SAM.

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.

Disclosure of Financial Relationships
As an accredited provider of the ACCME, SCA adheres to all ACCME Standards for Integrity and Independence in Accredited Continuing Education. The following individuals in control of content development for this activity have indicated that they do have financial relationships with ACCME defined ineligible companies within the past 24 months. All financial relationships have been mitigated. All have indicated that they have no financial relationships to disclose.

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1. Go to **https://scauniversity.pathlms.com/courses/90394**
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3. Print all pages of your certificate for your records.

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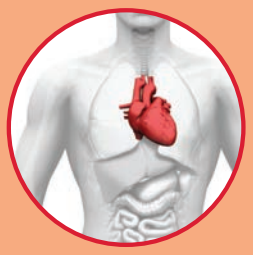

ECHO CME

TEE Evaluation Post Mitral Valve Repair

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CASE PRESENTATION

The patient is an 81-year-old female with HTN, HLD GERD, reflux esophagitis, hiatal hernia, and severe Type II posterior MR who was scheduled to undergo robotic mitral valve repair.

WATCH VIDEO 1

Upon completion of the mitral valve repair, the following image is found.

WATCH VIDEO 2

Question 1

What is most concerning?

a. Concern for VSD
b. Poor repair of the Mitral valve
c. Presence of systolic anterior motion of mitral valve (SAM)
d. Concern for newly wall motion abnormality not seen previously

CASE PRESENTATION

After separating from CPB, SAM was noted.

Question 2

The surgeon enquires if there is a way to predict this risk of SAM on preoperative TEE. Yes, there is! With mitral valve repairs, certain findings are associated with increased SAM risk post MV repair. Which of the following is not associated with an increased risk of SAM?

a. AL/PL ratio >3
b. Aorto Mitral angle <90
c. C-Sept of 1.1
d. Small left ventricle < 34mm

Question 3

When inspecting the new repair and the newly noted SAM which of the following is an appropriate communication to the surgeon?

a. It seems reasonable to proceed with closure.
b. Sorry Dr. Mitra Valve Surgeon but there is SAM we need to get back on bypass right away.
c. I am going to start dobutamine at 5mcg/kg/min to help with function as we come off pump.
d. Do you think we can fill the heart a bit and let's get the MAPs higher so I can better assess the degree of SAM?

Question 4

After optimizing the hemodynamics SAM persisted requiring surgical correction. Which of the following is not a possible correction used

a. Alfieri stitch
b. Shortened neochord
c. Downsizing the annuloplasty band
d. Septal myectomy

WATCH VIDEO 3

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ANSWERS

Answer 1

C. Presence of systolic anterior motion of mitral valve (SAM)

- It is clear there is turbulent flow in the LVOT with color flow. It is apparent the LVOT becomes obstructed by the mitral valve. This is a possible problem following mitral valve repair that one needs to stay vigilant for following separation from CPB.

Answer 2

A. AL/PL ratio >3

- When performing a mitral valve repair it is important to asses for risk of SAM prior to surgical intervention. This will assist the surgeon in the patient's risk for SAM following the repair. If a patient has a CSEPT <2.5 cm, a small left ventricle < 34mm, an aortic mitral angle <120, and an AL/PL <1 they would be considered high risk for SAM following MVR.

Answer 3

D. Do you think we can fill the heart a bit and let's get the MAPs higher so I can better assess the degree of SAM?

- Although it is true that if SAM is seen it is important to notify the surgeon, returning to CPB immediately is not always the correct answer. When managing post-repair SAM there are two options; medical management vs surgical correction. First, you will optimize the patients hemodynamics to assess the SAM. While in the OR you can increase their preload, increase their afterload, avoid tachycardia, and avoid inotropic agents. If the SAM has low gradients and MR is mild it is reasonable to proceed toward closure with medical management. If they continue to have > mild MR or high gradients then it is recommended to return to CPB and surgically correct it.

Answer 4

C. Downsizing the annuloplasty band

- There are a few ways that surgical correction could help SAM following mitral valve repair. The large majority of the interventions work on pulling the coaptation point posteriorly so there is nothing obstructing the LVOT anteriorly. This can be done by shortening the posterior leaflet or using shortened neochords to tac the posterior leaflet back. Another intervention could be using an Alfieri stitch which sutures the P2 and A2 cusp together pulling the anterior leaflet out of the LVOT or upsizing the annuloplasty band. The final intervention would be a septal myectomy. This option should be used with great caution and by a skilled surgeon as the risk of VSD formation is ever present.

References

1. Ashikhmina E, Schaff HV, Daly RC, Stulak JM, Greason KL, Michelena HI, Fatima B, Lahr BD, Dearani JA. Risk factors and progression of systolic anterior motion after mitral valve repair. J Thorac Cardiovasc Surg. 2021 Aug;162(2):567-577. doi: 10.1016/j.jtcvs.2019.12.106. PMID: 32173099.
2. Charls LM. SAM-systolic anterior motion of the anterior mitral valve leaflet post-surgical mitral valve repair. Heart Lung. 2003 Nov-Dec;32(6):402-6. doi: 10.1016/j.hrtlng.2003.07.004. PMID: 14652532.
3. Varghese R, Anyanwu AC, Itagaki S, Milla F, Castillo J, Adams DH. Management of systolic anterior motion after mitral valve repair: an algorithm. J Thorac Cardiovasc Surg. 2012 Apr;143(4 Suppl):S2-7. doi: 10.1016/j.jtcvs.2012.01.063. PMID: 22423603.
4. Alfieri O, Lapenna E. Systolic anterior motion after mitral valve repair: where do we stand in 2015? Eur J Cardiothorac Surg. 2015 Sep;48(3):344-6. doi: 10.1093/ejcts/ezv230. 4. PMID: 26142473.

LITERATURE REVIEW

Association Between Inspired Oxygen Fraction and Development of Postoperative Pulmonary Complications in Thoracic Surgery: A Multicentre Retrospective Cohort Study

Nicholas J. Douville, Douglas A. Colquhoun, Mark E. Smolkin, Bhiken I. Naik, Michael R. Mathis, Sachin Kheterpal, Stephen R. Collins, Linda W. Martin, Wanda M. Popescu, Nathan L. Pace, Randal S. Blank, on behalf of the Multicentre Perioperative Clinical Research Committeey

British Journal of Anaesthesia, 133 (5): 1073e1084 (2024)
doi: 10.1016/j.bja.2024.08.005

Reviewer:
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Baylor Scott and White Health

Background

There is not enough conclusive evidence to guide management of FiO2 during one lung ventilation in thoracic surgery. Some large observational studies of surgical patients have associated alveolar hyperoxia with increased mortality, postoperative ARDS and postoperative pulmonary complications.[1,2,3] However, recent meta-analysis in surgical patients failed to identify high alveolar FiO2 as a risk factor for postoperative pulmonary complications.[4,5]

The authors of this study examined whether there is a relationship between exposure to high concentrations of inspired oxygen (FiO2 >80%) during one lung ventilation for lung resection surgery and postoperative pulmonary complications.

Methods

This is a retrospective multicenter cohort study utilizing data from two large databases, the Multicenter Perioperative outcomes Group (MPOG)[6] and the Society of Thoracic Surgeons General Thoracic Surgery Databases (STS-GTSD),[7] with integration using patient level identifiers, as described before.[8] Study approval was obtained from the University of Virginia Review Board. Data collection methods, outcomes and statistical analyses were reviewed in advance and approved by the peer review committee of Multicenter Perioperative Outcomes Group (MPOG), 7/13/2020.[9]

Inclusion criteria: Adults (≥18 yr) who underwent lung resection surgery, assisted with one lung ventilation for ≥ 60 minutes, between 2012-2020.

Exclusion criteria: Patients who required one lung ventilation the previous 90 days, lung resection surgery requiring cardiopulmonary bypass or ECMO, lung transplantation or pneumonectomy.

The study design, inclusion and exclusion criteria allowed capture of data from a wide variety of oxygenation practices, surgical techniques and several lung resection procedures of different duration.

The primary outcome was postoperative pulmonary complications, possibly related to high FiO2 exposure during one lung ventilation for lung resection, including pneumonia, atelectasis, ARDS, respiratory failure, reintubation and need for ventilatory support for >48 h. A post hoc sensitivity analysis was done excluding "atelectasis" because of its different and variable spectrum of clinical significance and outcomes. As secondary outcomes, non-pulmonary post operative complications, including death were captured.

Exposure of interest was FiO2 above 80% expressed as area under the curve (AUC of FiO2 >80). Time -discretionary high FiO2 (FiO2 >80% while spO2 >98%) and time weighted average were tested in sensitivity analyses.

In the statistical analyses, the outcomes were evaluated for the duration of the mechanical ventilation as well as for the duration of the exposure.

In the statistical model, FEV1 was included as a possible covariate as well as the missing FEV1 (not random effect) in the datasets.

Patient characteristics, anesthetic and surgical data, comorbidities and other covariates were extracted from the databases and analyzed.

Results

From 16028 cases screened, 2733 met criteria to be included in the statistical analysis. In 60% of

cases FiO2 >80 was used. Postoperative pulmonary complications were observed in 141 (5.2%), major morbidity in 854 (31.3%) and death within 30 days from surgery in 17 (0.6%).

Univariate analysis and Multivariate regression analysis showed a higher incidence of postoperative pulmonary complications with high FiO2 exposure. Because the AUC does not only incorporate the impact of FiO2 but also the duration of ventilation, sensitivity models were designed to better clarify the results. It was shown that the duration of ventilation and not the high average FiO2 was associated with postoperative pulmonary complications, and that discretionary high FiO2 had no significant association with the primary outcome.

Discussion

Data analysis of this cohort does not support lower FiO2 to avoid pulmonary complications. The results are in line with previous studies which showed that the duration of mechanical ventilation and one lung ventilation are associated with adverse pulmonary outcomes.[10,11] However they are not in line with current practice and guidelines, which is to use the lowest possible FiO2 that achieves adequate oxygenation. There is concern of direct oxygen toxicity to the lung tissue,[12] increase in pulmonary capillary permeability13 and oxidative stress during lung surgery.[14] From the results of this study, it appears that there is an opportunity to decrease the FiO2 further and underscores that adverse post operative pulmonary outcomes are also related to the prior lung condition and the duration of one lung ventilation. Additional studies are needed.

Study LImitations

The retrospective nature of the study.
Significant number of patients were excluded due to missing information/ data not available.

References

1. Staehr-Rye AK, Meyhoff CS, Scheffenbichler FT, et al. High intraoperative inspiratory oxygen fraction and risk of major respiratory complications. Br J Anaesth 2017; 119:140-9.
2. McIlroy DR, Shotwell MS, Lopez MG, et al. Oxygen administration during surgery and postoperative organ injury: observational cohort study. BMJ 2022; 379, e070941.
3. Blum JM, Stentz MJ, Dechert R, et al. Preoperative and intraoperative predictors of postoperative acute respira tory distress syndrome in a general surgical population. Anesthesiology 2013; 118:19-29.
4. Wetterslev J, Meyhoff CS, Jørgensen LN, Gluud C, Lindschou J, Rasmussen LS. The effects of high perioper ative inspiratory oxygen fraction for adult surgical pa tients. Cochrane Database Syst Rev 2015, CD008884.
5. Mattishent K, Thavarajah M, Sinha A, et al. Safety of 80% vs 30-35% fraction of inspired oxygen in patients undergoing surgery: a systematic review and meta-analysis. Br J Anaesth 2019; 122: 311-24
6. Colquhoun DA, Shanks AM, Kapeles SR, etal. Considerations for integration of perioperative electronic health records across institutions for research and quality improvement: the approach taken by the Multicenter Perioperative Outcomes Group. Anesth Analg 2020; 130: 1133-46
7. Shahian DM, Jacobs JP, Edwards FH, et al. The society of thoracic Surgeons national database. Heart 2013; 99: 1494-501
8. Colquhoun DA, Leis AM, Shanks AM, et al. A lower tidal volume regimen during one-lung ventilation for lung resection surgery is not associated with reduced post operative pulmonary complications. Anesthesiology 2021; 134:562-76
9. Perioperative Clinical Research Committee (PCRC). Available from https://mpog.org/events/perioperative-clinical research-committee-pcrc/(accessed 16 June 2021).
10. Lai G, Guo N,Jiang Y, Lai J, Li Y, Lai R. Duration of one-lung ventilation as a risk factor for postoperative pulmonary complications after McKeown esophagectomy. Tumori 2020; 106:47-54
11. Licker M, de Perrot M, Spiliopoulos A, et al. Risk factors for acute lung injury after thoracic surgery for lung cancer. Anesth Analg 2003; 97: 1558-65
12. Heerdt PM, Stowe DF. Single-lung ventilation and oxida tive stress: a different perspective on a common practice. Curr Opin Anaesthesiol 2017; 30:42-9
13. Royer F, Martin DJ, Benchetrit G, Grimbert FA. Increase in pulmonary capillary permeability in dogs exposed to 100% O2. J Appl Physiol 1988; 65: 1140-6
14. Williams EA, Quinlan GJ, Goldstraw P, Gothard JW, Evans TW. Postoperative lung injury and oxidative dam age in patients undergoing pulmonary resection. Eur Respir J 1998; 11: 1028-34

Clinical Implications of Left Ventricular Apex Mechanics in Patients with Apical Hypertrophic Cardiomyopathy

Reviewer:
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Strengths

1. **Methodology:** The study is well-structured, analyzing 104 patients divided into subtypes based on apical maximum wall thickness and hypertrophy extent. The use of 2D STE for strain and twist measurements provides robust and detailed insights into myocardial mechanics.
2. **Findings:**
   - Even early stages of ApHCM demonstrate apical strain impairments while preserving apical rotation.
   - Advanced stages show further deterioration, including abnormal apical rotation and reductions in global LV twist, correlating with clinical symptoms and functional impairment.
3. **Clinical Relevance:** Apical rotation emerges as an independent predictor of adverse events such as atrial fibrillation, heart failure hospitalization, and the need for myectomy. This highlights its utility as a prognostic marker.
4. **Statistical Rigor:** The study employs logistic regression and Cox proportional hazards models, demonstrating significant associations between apical mechanics and clinical outcomes.

LImitations

1. **Cohort Size and Follow-up:** The single-center design and relatively small sample size may limit generalizability. Additionally, a median follow-up of 26 months restricts long-term prognostic insights.
2. **Imaging Constraints:** While 2D STE provides valuable data, the lack of three-dimensional imaging could limit the spatial accuracy of strain measurements.
3. **Demographic Bias:** Predominantly male participants (80%) may skew findings and limit applicability to females, who may present different hypertrophic patterns and risks.
4. **Limited Imaging Modality:** Only 27% of participants underwent cardiac MRI, reducing data granularity regarding fibrosis and ischemia

Conclusion

This article makes a significant contribution to understanding ApHCM pathophysiology and progression. It emphasizes the role of LV apex mechanics as both diagnostic and prognostic tools, suggesting that apical rotation could aid in early identification and risk stratification. However, further multicenter studies with larger sample sizes and longer follow-up periods are needed to validate these findings and refine clinical decision-making processes.

Dobutamine Stress Echocardiography in Low-Gradient Aortic Stenosis

Reviewer:

Richard Thalappillil, MD
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Tufts Medical Center

Background

Dobutamine stress echocardiography (DSE) is recommended to distinguish between true severe and pseudo-severe aortic stenosis (AS) in patients with reduced stroke volume and left ventricular ejection fraction (LVEF) <50% in the setting of low valve gradients. In addition, aortic valve (AV) calcification as assessed by cardiac computed tomography (CT) is another method to discriminate moderate and severe AS when gradients are low.

Methods

This prospective observational study included patients with low-gradient AS (mean gradient <40 mmHg and aortic valve area (AVA) <1.0 cm2) and low stroke volume (SV) (indexed SV ≤35 mL/m2) from three patient cohorts in Canada and Denmark between 2019 and 2022. Patients were excluded for missing data or other significant valvulopathy. All patients underwent comprehensive transthoracic echocardiogram, DSE, and cardiac CT.

Results

556 patients met inclusion criteria and 221 patients had complete DSE and cardiac CT datasets. 78 patients (35%) had LVEF <35%, 67 patients (30%) with LVEF 35% to 50%, and 76 patients (34%) with LVEF >50%. Severe AS by AV calcification was present evenly in all three groups. DSE increased SV in all groups, although 56% still had low stroke volume. Lower EF patients had higher contractile reserve during DSE defined as an increase in indexed stroke volume >20%.

The optimal cutoff values for discriminating severe and pseudo-severe AS during DSE were mean gradient of 34 mmHg, Vmax of 3.89 m/s, and AVA 0.9 cm2 with a sensitivity/specificity of 75%/64% (LVEF <35), 70%/63% (LVEF 35-50%), and 57%/59% (LVEF >50%). If compared to guideline recommendations of mean gradient > 40 mmHg, Vmax > 4 m/s, and AVA < 1.0 cm2, the sensitivity/specificity was 49%/78% versus 64%/66%, versus 78%/40%, respectively. DSE had diagnostic heterogeneity to predict severe AS among all LVEF groups.

Discussion

This study attempted to identify the choices of DSE threshold gradients, given the confirmation of severe AS has been based on patient outcome or surgeon evaluation during surgery. This is problematic as even moderate AS can be associated with poorer patient outcomes in patients with LVEF <50%. There is a large proportion of patients with high AV calcification that are labeled pseudo-severe AS based on DSE in patients with LVEF <50%. The DSE parameters provided greatest diagnostic utility in patients with LVEF <35% and modest utility in LVEF >35%. The best cutoff was a mean gradient 30 mmHg patients in patients with LVEF <35% and 40 mmHg in those with LVEF>35%.

Conclusion

DSE is safe in patients of all LVEF and led to an increase in SV in all patients with low-gradient AS. The highest accuracy for DSE to distinguish pseudo-severe and severe AS is in LVEF <35%. There is heterogeneity in DSE results patients with LVEF ≥35% and optimal DSE cutoffs vary.

Electroencephalography-Guided Anesthesia and Delirium in Older Adults After Cardiac Surgery: The ENGAGES-Canada Randomized Clinical Trial

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Reviewer:

Margo Hoyler, MD
Weill Cornell Medical Center
New York, NY

Introduction

Postoperative delirium is common following cardiac surgery, occurring in up to 50% of cases.[1] It is associated with increased hospital and ICU duration, increased healthcare spending, and long-term clinical sequelae such as cognitive decline.[2] Established risk factors for postoperative delirium include advanced age, pre-existing cognitive impairment and patient frailty.[1,3] Electroencephalography (EEG) is a demonstrated tool for monitoring anesthetic depth, with electroencephalographic suppression an accepted as a biomarker of deep anesthesia.[4] Previous data suggest that excessive depth of anesthesia may also contribute to delirium risk, although research in the cardiac population is limited and inconclusive.[2,5] The purpose of the current study was to evaluate if EEG-guided anesthesia was associated with decreased risk of delirium in older adults undergoing cardiac surgery.

Methods

This study was a multicenter, evaluator- and patient-blinded randomized controlled trial, conduced at 4 tertiary care hospitals in Canada. Randomization was performed by site in a 1:1 fashion. Patients were randomized to EEG-guided anesthesia or usual care. Eligible patients were over 60 years of age, scheduled to undergo cardiac surgery with cardiopulmonary bypass. Exclusion criteria included preoperative delirium, sensory impairments, and previous intraoperative awareness. Recruitment occurred between December, 2016 and February 2022.

EEG-guided care involved the display of EEG waveforms and derived parameters (e.g suppression ratio and spectral edge frequency among others). Clinicians were encouraged to titrate volatile anesthetic to minimize EEG suppression.

The primary outcome was delirium incidence during postoperative days 1 to 5, as diagnosed by the Confusion Assessment Method (CAM or CAM-ICU). Secondary outcomes included median hospital and ICU length of stay (LOS) as well as adverse postoperative events and mortality. Exploratory analyses were performed to identify potential delirium risk factors.

Assuming a baseline delirium rate of 25%, a sample size of 1132 patients was required to detect a clinically meaningful reduction of 8% in the primary outcome, with alpha = 0.05 and 90% power.

Results

1225 patients were enrolled and 1140 were randomized (573 to usual care and 567 to the EEG intervention). Patient baseline characteristics were similar between groups, as were intraoperative intravenous medications including hypnotic and amnestic agents, opioids and neuromuscular blockers. Surgical parameters were also similar across groups, including duration of surgery, CPB and aortic cross-clamp. EEG-guidance was significantly associated with a 0.14 reduction (95% CI, −0.15 to −0.13) in median minimum MAC, and with a 7.7 minute reduction (95% CI, −10.6 to −4.7) in median total EEG suppression time. Study sites varied meaningfully in median volatile anesthetic administered and in duration of EEG suppression.

The primary outcome, postoperative delirium in days 1-5, occurred in 102 of 562 patients (18.15%) in the EEG-guided group and 103 of 569 (18.10%) in the usual care group (difference, 0.05% [95%

CI, −4.57% to 4.67%]). After covariate adjustment for baseline characteristics, EEG guidance was not associated with decreased odds of delirium (adjusted odds ratio, 1.12 [95% CI, 0.80-1.55]; P = .51). By contrast, older age, higher EuroScore II score, history of delirium, and history of depression were all associated with significantly increased adjusted odds of postoperative delirium. ICU and hospital LOS were similar across groups, but varied across study sites.

Delirium duration and severity, among other measures, were similar across groups. In both groups, patients with postoperative delirium had experienced significantly longer duration of EEG suppression. The estimated increased odds of postoperative delirium associated with EEG suppression was 4% (adjusted odds ratio, 1.04; [95% CI, 1.04-1.07]; P < .01) for every additional 5 minutes with EEG suppression.

There were no significant differences between groups in 30-day and 1-year mortality risk. No incidents of intraoperative awareness were reported.

Discussion

In the ENGAGES-Canada trial, EEG-guided volatile anesthesia, and an associated reduction in anesthetic depth and EEG suppression time, did not significantly reduce delirium incidence in older adults undergoing cardiac surgery with cardiopulmonary bypass.

The current findings are at odds with several studies showing that EEG-guided anesthesia, in particular, was associated with decreased delirium risk in high-risk, non-cardiac surgery patients.[2,6] By contrast, the results of the ENGAGES-Canada trial are consistent with research findings in non-cardiac surgery populations, such as hip fracture patients, in which no difference in delirium risk was shown according to anesthetic type (general versus spinal, with minimal or no sedation).[7] Most notably, the results of the ENGAGES-Canada study are similar to those of the preceding ENGAGES trial,[8] which applied analogous research methods to non-cardiac surgery patients and also found that EEG-guided anesthesia did not decrease the incidence of postoperative delirium.

The ENGAGES-Canada trial has notable strengths, including a robust methodology, redundancy in types of EEG monitors used, and multiple study sites. Although anesthesia practice varied across sites, including depth of anesthesia and time in EEG suppression, EEG guidance was associated with decreases in both parameters, without an associated reduction in delirium across study locations.

Limitations of the study include the possibility that delirium was under-detected post-operatively. The study also does not address the possibility that greater reductions in anesthetic depth and EEG suppression time may be required to affect delirium incidence, or that unevaluated subgroups (e.g. moderately or severely frail patients) may be more sensitive to reductions in anesthetic depth as pertains to delirium risk.[3] As a result, the current study does not "rule out" an association between anesthetic depth and postoperative delirium, or the possibility that EEG-guidance would be beneficial in specific populations.

Conclusion

The results of the ENGAGES-Canada trial, EEG-guidance to minimize suppression was not associated with a decrease in the primary outcome of postoperative delirium. These findings do not support the use of intraoperative EEG for the prevention of delirium in older cardiac surgery patients.

References

1. Gosselt AN, Slooter AJ, Boere PR, Zaal IJ. Risk factors for delirium after on-pump cardiac surgery: a systematic review. Crit Care. 2015;19(1):346. doi:10.1186/s13054-015-1060-0.
2. Evered LA, Chan MTV, Han R, et al. Anaesthetic depth and delirium after major surgery: a randomised clinical trial. Br J Anaesth. 2021;127(5):704-712. doi:10.1016/j.bja.2021.07.021.
3. Li HC, Wei YC, Hsu RB, et al. Surviving and Thriving 1 Year After Cardiac Surgery: Frailty and Delirium Matter. Ann Thorac Surg. 2021 May;111(5):1578-1584. doi: 10.1016/j.athoracsur.2020.07.015. Epub 2020 Sep 17. PMID: 32949611; PMCID: PMC7965789.

4. Pedemonte JC, Plummer GS, Chamadia S, et al. Electroencephalogram Burst-suppression during Cardiopulmonary Bypass in Elderly Patients Mediates Postoperative Delirium. Anesthesiology. 2020 Aug;133(2):280-292. doi: 10.1097/ALN.0000000000003328. PMID: 32349072; PMCID: PMC7365754.
5. Ramachandran RV, Behera A, Hussain Z, et al. Incidence of Concurrent Cerebral Desaturation and Electroencephalographic Burst Suppression in Cardiac Surgery Patients. Anesth Analg. 2024 Oct 24. doi: 10.1213/ANE.0000000000007209. Epub ahead of print. PMID: 39446661.
6. Chan MTV, Cheng BCP, Lee TMC, Gin T; CODA Trial Group. BIS-guided anesthesia decreases postoperative delirium and cognitive decline. J Neurosurg Anesthesiol. 2013;25(1):33-42. doi:10.1097/ANA.0b013e3182712fba
7. Li T, Li J, Yuan L, et al; RAGA Study Investigators. Effect of regional vs general anesthesia on incidence of postoperative delirium in older patients undergoing hip fracture surgery: the RAGA randomized trial. JAMA. 2022;327(1):50-58. doi:10.1001/jama.2021.22647
8. Wildes TS, Mickle AM, Ben Abdallah A, et al; ENGAGES Research Group. Effect of electroencephalography-guided anesthetic administration on postoperative delirium among older adults undergoing major surgery: the ENGAGES randomized clinical trial. JAMA. 2019;321(5):473-483.

Effect of Preoperative Clopidogrel on Outcomes of Isolated Coronary Artery Bypass Graft: An STS National Database Analysis

Choi K, Schaff HV, Villavicencio MA, et al.
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Background

Dual antiplatelet therapy (DAPT) remains the standard of care for patients experiencing acute coronary syndrome (ACS) requiring percutaneous coronary intervention (PCI).[1] Studies comparing P2Y12 inhibitors such as the PLATO (Platelet Inhibition and Patient Outcomes) trial and the TRITON-TIMI 38 (Therapeutic Outcomes by Optimizing Platelet Inhibition With Prasugrel–Thrombolysis In Myocardial Infarction 38) study found that newer agents, ticagrelor and prasugrel, displayed no net benefit over the first PY212 inhibitor, clopidogrel, as the newer agents increased risk of major bleeding.[2-4] The bleeding risk associated with DAPT is especially concerning in the perioperative period for patients presenting for cardiac surgery, hence why guidelines recommend holding P2Y12 agents prior to cardiac surgery.

According to the national Society of Thoracic Surgeons Adult Cardiac Surgery Database (STS-ACSD) the prevalence of patients undergoing isolated coronary artery bypass grafting (CAPG) after PCI has increased by nearly 30% between 2008 (21%) and 2018 (29%).[5] Such an increase merits further investigation into optimal guidelines regarding appropriate timing of clopidogrel discontinuation before isolated CABG surgeries. Prior studies, such as a meta-analysis investigating the optimal timing of clopidogrel discontinuation in ACS patients before CABG surgery, found that patients who discontinued clopidogrel >5 days before CABG had a lower incidence of reoperation and major bleeding, as well as a lower incidence in composite endpoints such as mortality, myocardial infarction, recurrent ischemia, stroke, and emergent revascularization.[6] However, this analysis categorized patients into only two broad groups: those who discontinued clopidogrel <5 days versus >5 days before CABG surgery. Further stratification within the group <5 days was not assessed and leaves room for speculation. There is still much to elucidate regarding recommendations for timing of clopidogrel discontinuation 0-5 days prior to CABG surgery. In this STS-ACSD analysis, Choi et al aimed to further stratify patients who discontinued clopidogrel between 0 and 5 days prior to CABG surgeries.

Methods

Choi et al retrospectively analyzed data from the national STS-ACSD on patients (n=148,317) who underwent primary isolated primary CABG after clopidogrel use between January and December 2008. Exclusion criteria included those who received vitamin K antagonists or glycoprotein inhibitors within 24 hours prior to CABG and those without any reported data on clopidogrel use. Patients who received clopidogrel <5 days prior to CABG were stratified by day of discontinuation (days 0-5) and compared to the control group, which included patients who received clopidogrel >5 days prior to CABG. The primary outcome was operative mortality (death within 30 days from surgery or before hospital discharge) and the secondary outcomes included mediastinal re-exploration for bleeding, blood product use (overall, intraoperative, and postoperative), and total, preoperative, and postoperative lengths of stay. Univariate comparisons were conducted using Pearson chi-squared tests for categorical data and 2-sample t-tests for continuous data. The adjusted odds ratios of the above study outcomes were examined based on number of days since last clopidogrel administration. Statistical significance was characterized by P values <.05.

Results

Of the 148,317 patients who underwent isolated CABG, 13.2% (19,553 patients) received clopidogrel withing first days prior to surgery. This group was further stratified by days from administration to surgery: 0 days in 2,978 patients (15.9%), 1 day in 4572 patients (24.4%), 2 days in 3,547 patients (19.0%), 3 days in 2,976 patients (15.9%), 4 days in 2,795 patients (14.9%), and 5 days in 1,834 patients (9.%). The remaining 128,764 patients took clopidogrel >5 days prior to surgery.

The authors found that the primary outcome of operative mortality was statistically significant in patients who received clopidogrel within 5 days of surgery (2.8% vs. 2.1%, P <.001) and that this risk was inversely proportional to time since last administration. However, multivariate analysis revealed the adjusted risk of operative mortality on days 1 through 5 was not increased from that of patients in the control group. Rather, only CABG on day 0 of administration was associated with an increased risk of mortality (OR, 1.39, 95% CI, 1.11-1.73; P= .003).

Additionally, the rate of mediastinal exploration was significantly higher in patients who received clopidogrel within 5 days of surgery (3.5% vs. 2.1%, P <.001) with the rate declining as day from last administration increased. This rate plateaued after day 3 (day 0, 4.7%; day 1 3.8%; day 2, 3.4%; day 3, 2.9%; day 4, 3.0%; day 5, 2.9%). Similarly, patients who received clopidogrel within 5 days of surgery had a higher overall blood product utilization (72.7% vs. 56.8%, P <.001) with higher transfusion requirements correlating with a shorter time since last administration. A detailed breakdown revealed that patients who underwent surgery on day 0 required the highest volume of transfusions (79.7%), while the rates gradually declined over time, reaching 64.9% on day 5.

Postoperative complications, including prolonged ventilation (33.1% vs. 26.4%, P < .001), renal failure (3.9% vs. 3.4%, P < .001), and stroke (1.4% vs. 1.1%, P < .001), were more frequently observed in the clopidogrel group compared to the control group. Notably, prolonged ventilation was most common in patients who underwent surgery on day 0 (44.8%).

Discussion

This study identifies relevant clinical outcome data among patients taking clopidogrel in the immediate perioperative period (<5 days) prior to isolated CABG compared to patients who did not and further stratifies those patients by individual day of administration. The most striking findings in this study are evident in the direct comparison of clinical outcomes between patients who held clopidogrel for 3 versus 5 days prior to surgery, as it suggests that overall clinical outcomes between these patients were similar and even demonstrates that those in the day 3 group had a shorter total hospital length of stay.

Ultimately, despite the American College of Cardiology and American Heart Association guidelines recommending a five-day waiting period after discontinuing clopidogrel before performing CABG, this study suggests that surgery conducted after three days may provide comparable outcomes in terms of mortality and bleeding risks while potentially reducing hospital stays and healthcare costs and improving patient satisfaction.[7] The increased blood product utilization observed at day 3 compared to day 5 may present a trade-off for earlier surgical intervention, but the overall benefits in terms of efficiency and resource management could be significant.

However, as acknowledged by the authors, the retrospective nature of the study introduces potential limitations, including selection bias, as the timing of surgery was determined by individual clinical decisions. Additionally, the lack of granular data on the type and dosage of antiplatelet agents limits the ability to generalize findings across different ADP receptor inhibitors.

In conclusion, this large-scale observational study provides critical insights into the timing of CABG following clopidogrel discontinuation. While risks associated with early surgery remain modest, the findings suggest that performing CABG three days after discontinuing clopidogrel may offer a safe and efficient alternative to the traditionally recommended five-day waiting period, with no significant increase in operative mortality or re-exploration for bleeding.

References

1. Levine GN, Kern MJ, Berger PB, Brown DL, Klein LW, Kereiakes DJ, Sanborn TA, Jacobs AK; American Heart Association Diagnostic and Interventional Catheterization Committee and Council on Clinical Cardiology. Management of patients undergoing percutaneous coronary revascularization. Ann Intern Med. 2003 Jul 15;139(2):123-36. doi: 10.7326/0003-4819-139-2-200307150-00012. PMID: 12859162.
2. Wallentin L, Becker RC, Budaj A, et al. Ticagrelor versus clopidogrel in patients with acute coronary syndromes. N Engl J Med. 2009;361:1045–57.
3. Wiviott SD, Braunwald E, McCabe CH, et al. Prasugrel versus clopidogrel in patients with acute coronary syndromes. N Engl J Med. 2007;357:2001–15.
4. Koski R, Kennedy B. Comparative Review of Oral P2Y12 Inhibitors. P T. 2018 Jun;43(6):352-357. PMID: 29896034; PMCID: PMC5969212.
5. O'Brien SM, Feng L, He X, Xian Y, Jacobs JP, Badhwar V, Kurlansky PA, Furnary AP, Cleveland JC Jr, Lobdell KW, Vassileva C, Wyler von Ballmoos MC, Thourani VH, Rankin JS, Edgerton JR, D'Agostino RS, Desai ND, Edwards FH, Shahian DM. The Society of Thoracic Surgeons 2018 Adult Cardiac Surgery Risk Models: Part 2-Statistical Methods and Results. Ann Thorac Surg. 2018 May;105(5):1419-1428. doi: 10.1016/j.athoracsur.2018.03.003. Epub 2018 Mar 22. PMID: 29577924.
6. Should clopidogrel be discontinued before coronary artery bypass grafting for patients with acute coronary syndrome? A systematic review and meta-analysis Cao, Christopher et al. The Journal of Thoracic and Cardiovascular Surgery, Volume 148, Issue 6, 3092 - 3098
7. Lawton JS, Tamis-Holland JE, Bangalore S, et al. 2021 ACC/AHA/SCAI Guideline for Coronary Artery Revascularization: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. J Am Coll Cardiol. 2022;79:e21-e129.

Catheter Ablation or Antiarrhythmic Drugs for Ventricular Tachycardia — (VANISH2 Study)

What does this trial tell us that is new?

The VANISH2 trial demonstrated that in patients with ischemic cardiomyopathy and recurrent ventricular tachycardia, first-line therapy with catheter ablation as opposed to antiarrhythmic therapy, resulted in fewer VT episodes and ICD mediated shocks or medical intervention.

Sapp JL, Tang ASL, Parkash R, Stevenson WG, Healey JS, Gula LJ, Nair GM, Essebag V, Rivard L, Roux JF, Nery PB, Sarrazin JF, Amit G, Raymond JM, Deyell M, Lane C, Sacher F, de Chillou C, Kuriachan V, AbdelWahab A, Nault I, Dyrda K, Wilton S, Jolly U, Kanagasundram A, Wells GA; VANISH2 Study Team. Catheter Ablation or Antiarrhythmic Drugs for Ventricular Tachycardia. N Engl J Med. 2024 Nov 16. doi: 10.1056/NEJMoa2409501. PMID: 39555820.

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Background

In the first VANISH study, in 2016 (N Engl J Med 2016;375:111-121, DOI: 10.1056/NEJMoa1513614), we learned that in patients with ischemic cardiomyopathy with recurrent VT and an ICD, despite first line anti arrhythmic therapy, there was a lower rate of the composite primary outcome of death, VT storm or ICD shocks among patients who had a catheter ablation than among the patients receiving escalating antiarrhythmic therapy.

In VANISH2, described here, catheter ablation was compared to antiarrhythmic drug therapy as a first-line therapy for recurrent VT in ischemic cardiomyopathy.

Methods

This was an international, randomized and open-label trial. Patients with VT and prior MI with ischemic cardiomyopathy (416 patients) were randomized, 1:1, to either undergo catheter ablation (203 patients) or antiarrhythmic drug therapy (213 patients) with sotalol or amiodarone. These patients were followed for an average of 4.3 years.

Inclusion Criteria:

1. 18 years of age or older
2. History of prior myocardial infarction
3. Greater than or equal to one of the following events in the prior 6 month period off antiarrhythmic drug therapy

    a. Sustained monomorphic VT which ceased after cardioversion by drug or electricity, more than 3 VT symptomatic episodes treated with antitachycardia pacing, 5 or more VT events treated with antitachycardia pacing, or 1 or more ICD shocks or 3 or more VT events in 24 hours.

Exclusion criteria:

1. Active myocardial ischemia
2. Acute coronary syndrome in the 30 days prior
3. PCI less than 30 days or coronary artery bypass grafting less than 90 days prior
4. Prior VT catheter ablation
5. LV thrombus
6. Mechanical aortic or mitral valve

Results

Primary Outcome: The primary outcome, composite of all-cause death, VT storm, ICD shock, or sustained VT below ICD detection rate requiring treatment, for catheter ablation vs. antiarrhythmic drug therapy was: 50.7% vs. 60.6% (hazard ratio [HR] 0.75, 95% CI 0.58-0.97), p = 0.03. As a review, a hazard ratio of 1 suggests no difference between the groups, whereas

a hazard ratio greater than 1 indicates a higher risk of the event in the group being compared (catheter ablation group), and a hazard ratio of less than 1 indicates a lower risk of the event (composite of all-cause death, VT storm, ICD shock or sustained VT) in the group being compared (catheter ablation group). In VANISH2, a hazard ratio of 0.75 (less than 1), suggests that there is a lower risk of the composite of all-cause death, VT storm, ICD shock or sustained VT in the catheter ablation group.

Secondary Outcomes (catheter ablation vs antiarrhythmic drug therapy)

1. All-cause death: 22.2% vs. 25.4% (HR 0.84, 95% CI 0.56-1.24)
2. Appropriate ICD shock: 29.6% vs. 38.0% (HR 0.75, 95% CI 0.53-1.04)
3. VT storm: 21.7% vs. 23.7% (HR 0.95, 95% CI 0.63-1.42)
4. Sustained VT below ICD detection rate requiring treatment: 4.4% vs. 16.4% (HR 0.26, 95% CI 0.13-0.55)

In each of the secondary outcomes, the hazard ratio is < 1 which suggests a lower risk of each of the secondary outcomes in the catheter ablation group.

Discussion

As demonstrated above, the first VANISH trial demonstrated a decreased incidence of recurrent VT events in patients already on antiarrhythmic drug treatment who received catheter ablation vs escalation of their antiarrhythmic drug treatment. VANISH2, for the first time, establishes the role of catheter ablation for ischemic VT as the initial treatment modality over traditional antiarrhythmic drug therapy. While the mortality overall remained high in both groups given the nature of the disease process, ICD shocks for sustained VT were less in the catheter ablation group. It is important to keep in mind that this data is specific to ischemic cardiomyopathy mediated VT and cannot be extended to nonischemic cardiomyopathy, which is a different entity in the sense that the scar tissue that is the substrate for VT may be endocardial or epicardial with multiple foci.

This study establishes that in patients who have not been on antiarrhythmic therapy, proceeding with catheter ablation as first-line therapy for ischemic VT may improve quality of life and reduce events, especially in patients at high risk for adverse events from antiarrhythmic drug therapy.

References

1. Sapp JL, Tang AS, Parkash R, et al., for the VANISH2 Study Team. Catheter Ablation or Antiarrhythmic Drugs for Ventricular Tachycardia. N Engl J Med 2024;Nov 16: [Epub ahead of print].

PRO CON

INTRODUCTION TO PRO CON DISCUSSION

Hemodynamics Assessment of Right Ventricular Function Echocardiography vs Right Heart Catheterization (Pulmonary Artery Catheterization)

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PRO/CON DEBATE

SOCIETY OF CARDIOVASCULAR ANESTHESIOLOGISTS
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Right ventricular (RV) dysfunction and failure is associated with significant morbidity and mortality.[1,2,3] While refractory RV failure is uncommon, reported mortality is as high as 75%.[4] Right heart function/dysfunction is a strong predictor of outcome perhaps even more significant than LVEF.[5,6,7,8,9,10] RV systolic dysfunction and its response to inotropes are predictors of a range of cardiothoracic adverse outcomes and mortality, making its assessment important for prognosis and treatment considerations.[4,7,10,11]

Most often RV failure is secondary to left heart failure and pulmonary hypertension. Primary RV failure occurs in 10% or less and RV failure due to pericardial pathology accounts for less than 1-2%.[12,13] The clinical picture of RV failure is the result of reduced left heart filling and output, and the result of backward pressure, venous congestion or venous hypertension. The combination impairs end organ perfusion and function.[14] Understanding the normal function of the right heart is important to understand management of RV failure.[15]

Whether primary or secondary RV failure is associated with higher morbidity and mortality.[7,16,17,18] RV failure is a clinical syndrome characterized by increased chamber pressure and volume. The increased right ventricular chamber pressure causes a leftward shift and flattening of the interventricular septum (IVS) impairing both diastolic and systolic performance thru ventricular interdependence.[19,20,21,22,23,24,25,26]

In the healthy state with normal hemodynamics, the right ventricle is a low-pressure chamber that ejects blood forward into a low resistance pulmonary vascular system. Under these conditions the pressure volume curve of the right ventricle does not have defined isovolumic phases with the appearance of a passive conduit.[27,28,29,30] Several experimental studies have demonstrated minimal or no hemodynamic deterioration with the either akinesis of the RV free wall, or replacement of it with a non-contractile, non-distensible patch.[31,32,33] In the extreme example, patients with Fontan anatomy and physiology, have no sub-pulmonic ventricle yet blood passes down a gradient from the cavae to the pulmonary arterial tree.[34,35,36]

When assessing the right heart or, more specifically the right ventricle, there are two basic principles: 1) Right Ventricular-Pulmonary Artery Coupling and 2) Ventricular Interdependence. Understanding coupling between the RV and pulmonary circulation, and the interaction or interdependence between the two ventricles explains how the RV can be passive or bypass-able at one time, but ultimately critical for hemodynamic stability.[20,21,37,38,39,40,41,42,43]

In the heart without congenital defects the right ventricle and pulmonary arterial tree are 'coupled' meaning that blood flows from the right heart the pulmonary arteries with minimal energy usage. Normally, a low mean pulmonary artery pressure (mPAP < 20mmHg) and low pulmonary vascular resistance (PVR; 50 dynes sec cm-5 to 180 dynes sec cm-5 or < 2 Wood Units) are easily overcome by higher generated pressure within the RV (dP/dt).[30,44] This pressure gradient generates pulmonary blood flow (QPA), which passes through the pulmonary vascular system to the left heart and out into the systemic circulation generating blood flow (cardiac output), a pressure gradient, and a perfusing pressure.

Assessing the ability of RV contractility (Elastance or recoil; Ees) to overcome resistance or PA afterload or elastance (Ea) (i.e. Coupling) is best measured during right heart catheterization.[41,42,44,46]

RV-PA Coupling = Ees/Ea
Ees = (PESP)/(RVESV)
PESP = Pulmonary end systolic pressure
RVESV = RV End Systolic Volume
Ea = PESP/SV
SV = Stroke volume

Single beat estimates of RV-PA coupling using SV/ESA during cardiac magnetic resonance imaging correlates with function and outcome.[45,46,47,48,49,50]

Ees/Ea = (PESP/RVESV)/(PESP/SV)
alternatively:
Ees/Ea = SV/RVESV or SV/ESA
ESA = end systolic area

An SV/ESA < 1.0 has been reported in patients with PHTN. An SV/ESA < 0.5 is associated with RVEF < 35%, clinical decompensation and greater mortality.[41,43,48,49,50] Bedside echocardiographic measures SV/ESV (or ESA), RV strain, longitudinal contraction (tricuspid annular plane systolic excursion; TAPSE), and PA systolic pressures (PASP) evaluate RV performance.[48]
A TAPSE/PASP > 0.6 is considered normal and associated with favorable outcome.[30,42,44,46,47,48]

TAPSE/PASP > 0.6; normal function
TAPSE = Tricuspid Annular Plane Systolic Excursion
PASP = Pulmonary Artery Systolic Pressure

A TAPSE/PASP < 0.3 is indicative of RV-PA un-coupling, morbidity and mortality.[46,47]
A TAPSE/PASP between 0.3 and 0.6 represents a transition and an opportunity to intervene and prevent further decompensation.

Ventricular interdependence involves systolic and diastolic interactions between the RV and LV. The right and left hearts are anatomically and physiologically linked.[24,25,26] Although the right ventricle is a larger chamber, during imaging it appears smaller. Anatomically, the right ventricle wrapped around the more symmetric LV with the interventricular septum in between and by the connecting free wall fibers on the outside.[25,26,37,38,4]4 Experimental data reveals that the LV is responsible for up to 65-70% of the RV dP/dt and QPA i.e. systolic ventricular interdependence.[23] The anatomy and shape of the RV, the position and shape of the interventricular septum, and the systolic performance of the LV are key toward systolic ventricular interdependence. With normal LV-RV pressure gradients the interventricular septum is convex toward the right and contracts to be more so.[24,25,26,51,52,53,54,55] Coupled with free wall fiber connection and contraction the volume in the right heart is compressed generating RV dP/dt and QPA.[37]

In the presence of RV failure and dilation, whether due to primary or secondary, the RV dP/dt is not adequate to overcome the PA resistance and the RV-PA become uncoupled. Chronically the RV compensates with hypertrophy, increased contractility, and increased chamber size, however, over time and/or in the acute state, the right heart fails, described by poorly contractility and RV dP/dT and reduced QPA.[30,44,45,56] The increased volume and pressure will cause a leftward shift in the interventricular septum, impairing or reducing left heart filling or preload (diastolic ventricular interdependence), which reduces left ventricular dP/dt. This will subsequently further impair right ventricular systolic performance, i.e. RV dP/dt and QPA (systolic ventricular interdependence).[19,57,58] The resulting reduced systemic blood flow and pressure reduces coronary flow causing greater myocardial dysfunction.[59] A downward spiral takes place.

Management of right heart failure is based on optimizing ventricular interdependence and achieving RV-PA coupling. This involves adjustment/manipulation of pressures, pressure gradients, blood flow, and chamber volumes and shape, all with the goal of returning the right and left ventricular anatomic relationship toward a normal state. This includes a normally positioned and functioning IVS that is convex toward the right ventricle. For the anesthesiologist and the critical care specialist available tools include echocardiography and invasive hemodynamic catheters. The following Pro-Con debates which of these tools is best to accurately assess, monitor, and guide management of right ventricular failure.

1 Subramani S, Sharma A, Arora L, Hanada S, Krishnan S, Ramakrishna H: Perioperative right ventricular dysfunction: Analysis of outcomes. J Cardiothorac Vasc Anesthesia 2021;36:309-320.

2 Towheed A, Sabbagh E, Gupta R et al: Right ventricular dysfunction and short-term outcomes following left-sided valvular surgery: An echocardiographic study. J Am Heart Assoc 2021;10:e016283. DOI: 10.1161/JAHA.120.016283

3 Bootsma IT, de Lange F, Koopmans M et al: Right Ventricular Function After Cardiac Surgery Is a Strong Independent Predictor for Long-Term Mortality. J Cardiothorac Vasc Anesth. 2017;31:1656-1662.

4 Kaul TK, Fields BL: Postoperative acute refractory right ventricular failure: incidence, pathogenesis, management and prognosis. Cardiovasc Surg 2000;8:1–9.

5 Ro SK, Sato K, Ijuin S, Sela D, Fior G, Heinsar S, Kim JY, Chan J, Nonaka H, Lin ACW, Bassi GL, Platts DG, Obonyo NG, Suen JY, Fraser JF. Assessment and diagnosis of right ventricular failure-retrospection and future directions. Front Cardiovasc Med. 2023 May 30;10:1030864. doi: 10.3389/fcvm.2023.1030864. PMID: 37324632; PMCID: PMC10268009.

6 Konstam MA, Kiernan MS, Bernstein D et al: Evaluation and management of right-sided heart failure. Circulation 2018;137:e578-e622.

7 Maslow AD, Regan MM, Panzica P, Heindel S, Mashikian J, Comunale ME: Precardiopulmonary bypass right ventricular function is associated with poor outcome after coronary artery bypass grafting in patients with severe left ventricular systolic dysfunction. Anesth Analg 2002;95:1507-1518.

8 Moledina S, Pandya B, Bartsota M, Mortensen KH, McMillan M, Quyam S, Taylor AM, Haworth SG, Schulze-Neick I, Muthurangu V. Prognostic significance of cardiac magnetic resonance imaging in children with pulmonary hypertension. Circ Cardiovasc Imaging. 2013 May 1;6(3):407-14. doi: 10.1161/CIRCIMAGING.112.000082. Epub 2013 Apr 9. PMID: 23572488.

9 de Groote P, Millaire A, Foucher-Hossein C, Nugue O, Marchandise X, Ducloux G, Lablanche JM: Right ventricular ejection fraction is an independent predictor of survival in patients with moderate heart failure. J Am Coll Cardiol 1998;32:948-954.

10 Gorcsan J 3rd, Murali S, Counihan PJ, Mandarino WA, Kormos RL. Right ventricular performance and contractile reserve in patients with severe heart failure. Assessment by pressure-area relations and association with outcome. Circulation. 1996 Dec 15;94(12):3190-7. doi: 10.1161/01.cir.94.12.3190. PMID: 8989128.

11 Tadic M, Pieske-Kraigher E, Cuspidi C, Morris DA, Burkhardt F, Baudisch A, Haßfeld S, Tschöpe C, Pieske B. Right ventricular strain in heart failure: Clinical perspective. Arch Cardiovasc Dis. 2017 Oct;110(10):562-571. doi: 10.1016/j.acvd.2017.05.002. Epub 2017 Jun 29. PMID: 28669483.

12 Padang R, Chandrashekar N, Indrabhinduwat M et al: Aetiology and outcomes of severe right ventricular dysfunction. Eur Heart J 2020;21:1273-1282.

13 Galie N, Humbert M, Vachiery J-L et al: 2015 ESC/ERS guidelines for the diagnosis and treatment of pulmonary hypertension. Eur Respir J 2015;46:903-975.

14 Chen L, Hong L, Ma A, Chen Y, Xiao Y, Jiang F, Huang R, Zhang C, Bu X, Ge Y, Zhou J: Intraoperative venous congestion rather than hypotension is associated with acute adverse kidney events after cardiac surgery: a retrospective cohort study British Journal of Anaesthesia 2022;128(5):785e795 doi: 10.1016/j.bja.2022.01.032

15 Vachiery J-L, Simmoneau G: Management of severe pulmonary arterial hypertension. Eur Respir Rev 2010;19:279-287. DOI: 10.1183/09059180.00008010

16 Haddad F, Hunt SA, Rosenthal DN, Murphy DJ: Right Ventricular Function in Cardiovascular Disease, Part I: Anatomy, Physiology, Aging, and Functional Assessment of the Right Ventricle. Circulation 2008;117:1436-1448.

17 Haddad F, Doyle R, Murphy DJ, Hunt SA: Right Ventricular Function in Cardiovascular Disease, Part II: Pathophysiology, Clinical Importance, and Management of Right Ventricular Failure. Circulation 2008;117:1717-1731.

18 Moazami N, Pasque MK, Moon MR, Herren RL, Bailey MS, Lawton JS, Damiano RJ Jr. Mechanical support for isolated right ventricular failure in patients after cardiotomy. J Heart Lung Transplant. 2004 Dec;23(12):1371-5. doi: 10.1016/j.healun.2003.09.022. PMID: 15607666.

19 Hoffman D, Sisto D, Frater RWM, Nikilic SD: Left-to-right ventricular interaction with a noncontracting right ventricle. J Thorac Cardiovasc Surg 1994;107:1496-1502.

20 Santamore WP, Dell'Italia LJ: Ventricular interdependence: significant left ventricular contributions to right ventricular systolic function. Prog Cardiovasc Dis. 1988;40:289–308.

21 Lin SS, Reynertson SI, Louie EK, Levitsky S : Right ventricular volume overload results in depression of left ventricular ejection fraction. Implications for the surgical management of tricuspid valve disease. Circulation 1994;90:II209-213.

22 Friedberg MK, Redington AN: Right versus left ventricular failure. Differences, similarities, and interactions. Circulation 2014;129:1033-1044.

23. Damiano RJ, Jr, La Follette P Jr, Cox JL, Lowe JE, Santamore WP: Significant left ventricular contribution to right ventricular systolic function. Am J Physiol 1991;261:H1514–H1524.

24 Saleh S, Liakopoulos OJ, Buckberg GD: The septal motor of biventricular function. European J Cardiothorac Surgery 2006;295:S126-S138.

25 Torrent-Guasp F, Kocica MJ, Corno AF et al: Towards new understanding of the heart structure and function. Eur J Cardiothorac Surg 2005;27:191-201.

26 Kocica MJ, Corno AF, Carreras-Costa F et al: The helical ventricular myocardial band: global, three-dimensional, functional architecture of the ventricular myocardium, European Journal of Cardio-Thoracic Surgery, Volume 2006;29:S21–S40. https://doi.org/10.1016/j.ejcts.2006.03.011

27 Redington A, Gray HH, Hodson ME, Rigby ML, Oldershaw PJ: Characterisation of the normal right ventricular pressure-volume relation by biplane angiography and simultaneous micro manometer pressure measurements. Br Heart J 1988;59:23-30.

28 Zhao H-W, Wu A-S, Liu Y et al: Assessment of right ventricular function by pressure-volume loops in off-pump coronary artery bypass surgery. Chin Med J 2008;121:932-938.

29 Maughan WL, Sagawa SK, Weisfeldt ML: Instantaneous pressure-volume relationship of the canine right ventricle. Circ Res 1979;44:309-315.

30 Naeijie R, Brimioulle S, Dewachter L: Biomechanics of the right ventricle in health and disease (2013 Grover Conference Series). Pulm Circ 2014;4:395-406.

31 Kagan A: Dynamic responses of the right ventricle following extensive damage by cauterization. Circulation 19525:816-823.

32 Sawatani S, Mandell C, Kusaba E: Ventricular performance following ablation and prosthetic replacement of right ventricular myocardium. Trans Am Soc Artif Intern Organs. 1974;20:629-636.

33 Starr I, Jeffers W, Meade Jr RH: The absence of conspicuous increments of venous pressure after severe damage to the right ventricle of the dog, with a discussion of the relation between clinical congestive failure and heart disease. Am Heart J 1953;26:291-301.

34 Fontan F, Baudet E: Surgical repair of tricuspid atresia. Thorax 1971;26:240-248.

35 Pundi KN, Johnson JN, Dearani JA et al: 40 year follow-up after the Fontan operation: Long-term outcomes of 1052 patients. J Am Coll Cardiol 2015;13:1700-1710.

36 Dennis M, Zannino D, du Plessis K et al: Clinical Outcomes in Adolescents and Adults After the Fontan Procedure. J Am Coll Cardiol. 2018;71:1009-1017.

37 Li KS, Santamore WP: Contribution of each wall to biventricular failure. Cardiovascular Research 1993;27:792-800.

38 Friedberg MK, Redington AN: Right versus left ventricular failure. Differences, similarities, and interactions. Circulation 2014;129:1033-1044.

39 Sanz J, Sanchez-Quintana D, Bossone E, Bogaard HJ, Naeije R: Anatomy, Function, and Dysfunction of the Right Ventricle. J Am Coll Cardiol 2019;73:1464-1482.

40 Maughan WL, Sagawa SK, Weisfeldt ML: Instantaneous pressure-volume relationship of the canine right ventricle. Circ Res 1979;44:309-315.

41 Vonk-Noordegraaf A, Chin KM, Haddad F et al: Pathophysiology of the right ventricle and of the pulmonary circulation in pulmonary hypertension: an update. Eur Respir J 2019;53:1801900 https://doi.org/10.1183/13993003.01900-2018

42 Vonk-Noordegraaf A, Westerhof BE, Westerhof N: The Relationship Between the Right Ventricle and its Load in Pulmonary Hypertension. J Am Coll Cardiol 2017;69:236-243.

43 Vonk-Noordegraaf A, Westerhof N: Describing right ventricular function. Eur J Respir 2013;41:1419-1423.

44 Naeije R, Badagliacca R: The overloaded right heart and ventricular interdependence. Cardiovasc Res 2017;113:1474-1485.

45 Maeder MT, Weber L, Weilenmann D, Haager PK, Joerg L, Taramasso M, Buser M, Ehl NF, Maisano F, Rickli H. Invasive Hemodynamic Staging Classification of Cardiac Damage in Patients With Aortic Stenosis Undergoing Valve Replacement. Can J Cardiol. 2020 Oct;36(10):1667-1674. doi: 10.1016/j.cjca.2020.02.004. Epub 2020 Feb 8. PMID: 32416065.

46 Tello K, Ghofrani HA, Heinze C et al: A simple echocardiographic estimate of right ventricular-arterial coupling to assess severity and outcome in pulmonary hypertension on chronic lung disease. Europ Resp J 2019;54 doi.org/10.1183/13993003.02435-2018

47 Tello K, Wan J, Dalmer A et al: Validation of the tricuspid annular plane systolic excursion/systolic pulmonary artery pressure ratio for the assessment of right ventricular-arterial coupling in severe pulmonary hypertension. Circ Cardiovas Imaging 2019;12. DOI:10.1161/CIRCIMAGING.119.009047

48 Todaro MC, Carerj S, Trifiro MP, Consolo G, Khandheria B: Echocardiographic evaluation of right ventricular-arterial coupling in pulmonary hypertension. Am J Cardiovasc Dis. 2020;10;272-283. SSN:2160-200X/AJCD0116854

49 Sanz J, Garcia-Alvarez A, Fernandez-Friera L et al: Right ventriculo-arterial coupling in pulmonary hypertension: a magnetic resonance study. Heart 2012;98:238-243. doi:10.1136/heartjnl-2011-300462

50 Vanderpool RR, Pinsky MR, Naeije R et al: Right ventricular-pulmonary arterial coupling predicts outcome in patients referred for pulmonary hypertension. Heart 2015;101:37-43. doi:10.1136/heartjnl-2014-306142.

51 Kovacs A, Laatos B, Merkely B: Right ventricular mechanical pattern in health and disease: beyond longitudinal shortening. Heart Failure Reviews 2019;24:511-520.

52 Buckberg G, Hoffman JIE: Right ventricular architecture responsible for mechanical performance: Unifying role of ventricular septum. J Thorac Cardiovasc Surg 2014;148:3166-3171.

53 Woodard JC, Chow E, Farrar DJ. Isolated ventricular systolic interaction during transient reductions in left ventricular pressure. Circ Res. 1992 May;70(5):944-51. doi: 10.1161/01.res.70.5.944. PMID: 1568303.

54 Kingma I, Tyberg JV, Smith ER: Effects of diastolic transseptal pressure gradient on ventricular septal position and motion. Circulation 1983;68:1304-1314.

55 Belenkie I, Grabrielle Horne S, Dani R, Sith ER, Tyberg JV: Effects of aortic constriction during experimental acute right ventricular pressure loading. Circulation 1995;92:546-554.

56 Fenely MP, Olsen CO, Glower DD, Rankin JS: Effect of acutely increased right ventricular afterload on work output from the left ventricle in conscious dogs. Systolic ventricular interaction. Circ Res 1989;65:135-145.

57 Danton MH, Byrne JG, Flores KQ, Laurence RG, Cohn LH, Aklog L: Modified Glenn connection for acutely ischemic right ventricular failure reverses secondary left ventricular dysfunction. J Thorac Cardiovasc Surg 2001;122:80-91.

58 Kingma I, Tyberg JV, Smith ER: Effects of diastolic transseptal pressure gradient on ventricular septal position and motion. Circulation 1983;68:1304-1314.

59 Crystal GJ, Pagel PS: Right ventricular perfusion. Physiology and clinical implications. Anesthesiology 2018;128:202-218

Right Heart Catheterization/Pulmonary Artery Catheterization is the Best Modality to Assess and Manage Right Heart/Right Ventricular Failure

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Hemodynamics relates to the flow of blood to the organs and tissues of the body, which is governed by physical forces in accordance with Poiseuille's law:

Blood flow = ΔPπr4/8ηl
ΔP is the pressure gradient across the vessel
r is the vessel radius
η is the viscosity
l is the vessel length

The purpose of the cardiovascular and pulmonary systems is to delivery oxygenated blood to the systemic circulation and end-organs. Subsequently, deoxygenated blood is returned for gas exchange and recirculation. Tissue perfusion is related to blood flow, pressures and pressure gradients. Blood flows from a high pressure to a lower pressure.

The left and right ventricles are physically and physiologically linked such that the RV is wrapped or stretched around the LV, with the two chambers connected by with the free wall fibers and IVS in between, the latter of which appears convex toward the RV, and becomes more so during ventricular systole.[1,2] As a result of this link, systolic and diastolic ventricular interdependence exists.[1,2] Left ventricular free wall and septal contraction helps to compresses the RV volume and is responsible for 40-65% of RV pressure generation (dP/dt) and pulmonary blood flow (QPA); i.e. systolic ventricular interdependence.[3,4,5] RV failure results reduces QPA and causes a leftward shift of the IVS, both of which reduces left heart preload and subsequent pressure generation (i.e. diastolic ventricular interdependence) which subsequently compromises systolic ventricular interdependence.[6,7,8] Systemic and coronary blood flow and pressure decline causing ventricular and end-organ ischemia and dysfunction. Right heart failure causes backflow of pressure and volume or venous congestion and venous hypertension which also impairs end-organ perfusion.[9,10,11] All considered right heart failure perpetuates into a downward spiral.

Right heart failure and poor right heart performance are not necessarily synonymous with reduced right ventricular ejection fraction (RVEF), or fractional area of contraction (RVFAC).[12,13,14,15] Under the right conditions stable hemodynamics are possible despite the absence of a normally contracting RV.[6,16,17,18] Right heart function and performance are defined by the generation of right ventricular pressure (RV dP/dt) and pulmonary blood flow (QPA).[12] The Fontan data shows that, under the right conditions, a subpulmonic ventricle is not necessary at all demonstrating that blood flow is based on pressure gradients.[19,20,21,22] Failure of Fontan patients also shows the critical importance of pulmonary vascular resistance and pressures with subsequent reductions in forward flow and increased venous congestion.[20]

Experimental and clinical data both demonstrate that control of loading conditions and optimization of ventricular interdependence permit the right ventricle to stay coupled with the pulmonary vasculature to generate adequate RV dP/dt, QPA, all while preventing backflow or high venous (CVP or RAP) pressures and congestion.[1,6,9,13,14,15,23,24,25,26] Although echocardiographic surrogates for assessing coupling are described, right heart catheterization and assessment of pressure-volume relationships at different ventricular volumes is the gold standard.[24,25,26,27,28] Perioperative monitoring, diagnosis and management of right heart failure

are critically important to patient care and outcome.[9,13,15,23,29,30] Anesthesiologists and intensivist have access to both invasive hemodynamic monitors and echocardiography. Although both modalities exist, echocardiographic assessment is intermittent unless the echocardiographer is constantly present, requiring multiple manipulations and adjustments to obtain an estimate of what would be considered a comprehensive hemodynamic assessment. It is not considered a routine monitor in the intensive care. By contrast invasive right heart catheterization, pulmonary artery catheter (PAC), permits continuous measurements and analyses so that all levels of caregivers have access and can use the data to monitor for, diagnose and manage right heart failure. The continuous display of pulmonary artery pressures, right ventricular pressure, right atrial or central venous pressures, and measures of cardiac output provides ample amounts of right heart performance data and allows calculation of systemic and pulmonary vascular resistances.[13,14,15,24,25,26,31,32] The minimal amount of catheter adjustment to obtain pulmonary capillary wedge trace (PCWP) can be accomplished by multiple levels of care. Depending on the hardware used, periodic or continuous monitoring or measurement of mixed venous oximetry and cardiac output are possible.[14,15,29,33] The PAC allows blood gas determinations from the vena cava, right atrium and pulmonary artery for assessing total oxygen balance, and evaluation of cardiac shunt.[14,15,33] In addition, specific PAC can assess RVEF, RV pressure and RV volumes.[29,34,35,36] Care based on invasive monitors can be algorithm based and goal directed.

Hemodynamic data obtained from right heart catheterization is the standard to determine RV-PA coupling and levels of compensation or decompensation.[13,14,22,24,25,26,32,33] This is gold standard for diagnosing and assessing the severity of and differentiating between the causes of pulmonary hypertension necessary to determine types of therapy.[13,14,32,33] (Table 1). From the RHC multiple measures and ratios are obtainable and used management of the surgical patient including perioperative optimization, determinations of type and timing of invasive procedures based on assessment of levels of compensation (Table 2).

Table 1: Differentiation between pre and post capillary, and combined pulmonary hypertension

	mPAP mmHg	PVR (WU) WU=80 dynes·sec·cm^{-5}	PCWP mmHg	TPG (mPAP-PCWP) mmHg	DPG (PADP-PCWP) mmHg
Normal	< 20	< 3 WU	< 15		
Pre-Capillary	≥ 20	> 3 WU	< 15	≥ 12	≥ 7
Post-Capillary	≥ 20	< 3 WU	≥ 15	< 12	< 7
Combined/ Mixed	≥ 20	> 3 WU	≥ 15	≥ 12	≥ 7

WU = wood units; PCWP = pulmonary capillary wedge pressure; TPG = transpulmonary pressure gradient; DPG = Diastolic pulmonary pressure gradient; mPAP = mean pulmonary artery pressure; PADP = pulmonary artery diastolic pressure

Table 2: Right heart hemodynamic data and right/left heart hemodynamic ratios to determine levels of decompensation

Compensation vs Decompensation

Hemodynamics	Normal/ Compensated	Abnormal/Transition	Decompensation
Central Venous Pressure (CVP) (mmHg)	< 8	8 - 15	> 15
CVP/PCWP	< 0.6	0.6 - 1.0	> 1.0
Pulmonary Artery Systolic Pressure (PASP) (mmHg)	< 40	40 - 60	> 60
Mean Pulmonary Artery Pressure (mPAP) (mmHg)	< 25	25 - 40	> 40
Cardiac Index (L/min/m2)	> 2.5	2.0 - 2.5	≤ 2.0
Mean Pulmonary Artery/Mean Arterial Pressure	<0.3	0.3 - 0.75	> 0.75
Pulmonary Vascular Resistance Index (WU/m2)	< 3	3-8	> 8
Pulmonary Vascular/Systemic Vascular Resistance	< 0.1	0.3 - 0.5	> 0.5
Pulmonary Artery Systolic/Systemic Systolic	< 1/3	1/3 - 2/3	> 2/3
Transpulmonary/Transsystemic Gradient	< 0.1	0.1 - 0.5	> 0.5
PAPi (PASP-PADP)/CVP	> 1.85 (LVAD)	< 1.85 (LVAD)	
Ees/Ea (RV-PA coupling RHC)	> 1.5; 1.0 - 1.5	0.7 - 1.0	< 0.7
Stroke Volume/End Systolic Volume	> 0.5	< 0.5	
Heart rate (beats/min)	< 100	> 100	
Rhythm	Sinus rhythm	Atrial tachyarrhythmias	

CVP = central venous pressure; PCWP = pulmonary capillary wedge pressure; TPG = transpulmonary pressure gradient; DPG = Diastolic pulmonary pressure gradient; mPAP = mean pulmonary artery pressure; PASP = pulmonary artery systolic pressure; PADP = pulmonary artery diastolic pressure; Ees/Ea = Coupling measure; RHC – Right heart catheterization

Comparisons of right sided and left sided atrial (or pulmonary capillary wedge pressure; PCWP) pressures and resistances can differentiate between left heart and right heart dysfunction necessary to direct management and monitor its impact.[37, 38] While echocardiography can visualize the position of the IVS and its shape (convex, flat, concave) the actual position and shape are determined by relative right and left pressures.[39,40,41] (Table 2). It makes sense then that management, assessment, and recovery are based on optimizing these pressures and resistances.[6,7,8,24,25,26,27,28,41]

Determining the cause of pulmonary hypertension is critical to guiding therapy. In patients with pulmonary hypertension mPAP and RAP, mPAP, and PVR correlate with disease progression and survival.[42,43] **Ivan et al** reported that PAPi, which reflects RV-arterial coupling may help identify risk for developing RV failure.[31] For patients with types I, III, and IV pulmonary arterial hypertension, hemodynamic measures including CVP, PAP and PVR guide management including pulmonary vasodilator therapy.[44] Management of PHTN and monitoring of therapeutic response is based on reductions of mPAP and PVR.[45,46,47,48,49]

Depending on the technology and methods, cardiac output, or pulmonary blood flow, can be determined continuously or intermittently by any caregiver. It is well known that low cardiac output syndrome (Cardiac index < 2L/min/m2) is associated with greater postoperative mortality in cardiac surgical patients.[14,50,51] Ultimately blood needs to move forward i.e. cardiac output. In one study the incidence was 9.1% and those with low cardiac output syndrome had a 17x greater mortality.[51] Using cardiac output data, pulmonary and systemic vascular resistances, stroke volume, and right ventricular stroke work can be calculated:[13,14,52,53]

SVR = (MAP – CVP)/CO
PVR = (mPAP – PCWP)/CO
RVSW = Stroke Volume x (mPAP – RAP)

Pressure assessment and monitoring and waveform analysis are part of the value and benefits of invasive monitoring in assessing right heart function.

Central Venous Pressure

The central venous pressure, or right atrial pressure, and waveform reflects upon right heart function.[54,55] An elevated RAP indicates backup of flow or venous congestion, or venous hypertension impairs end-organ perfusion and is associated with adverse outcome.[9,10,11] **Chen et al** reported that incremental increases in venous pressures are more significantly associated with post cardiac surgery renal dysfunction than systemic hypotension.[9]

In a matched cohort study comparing 2174 septic patients with CVP monitoring to 2174 septic patients without a CVP monitor, the use of CVP monitoring was associated with reductions in mechanical ventilation time, vasopressor use, lactate levels, and mortality.[55] Patients monitored with CVP received more fluid in the first 24 hours.[55] Patients with initial CVP < 8 mmHg had better outcomes.[55] In a more recent analysis, the magnitude and duration of an elevated CVP (> 10 mmHg) was predictive of 28-day mortality in septic patients.[56] Elsewhere in 1986 ICU patients a CVP > 10mmHg was associated with increased renal injury, length of ICU and hospital stays, and greater mortality.[57]

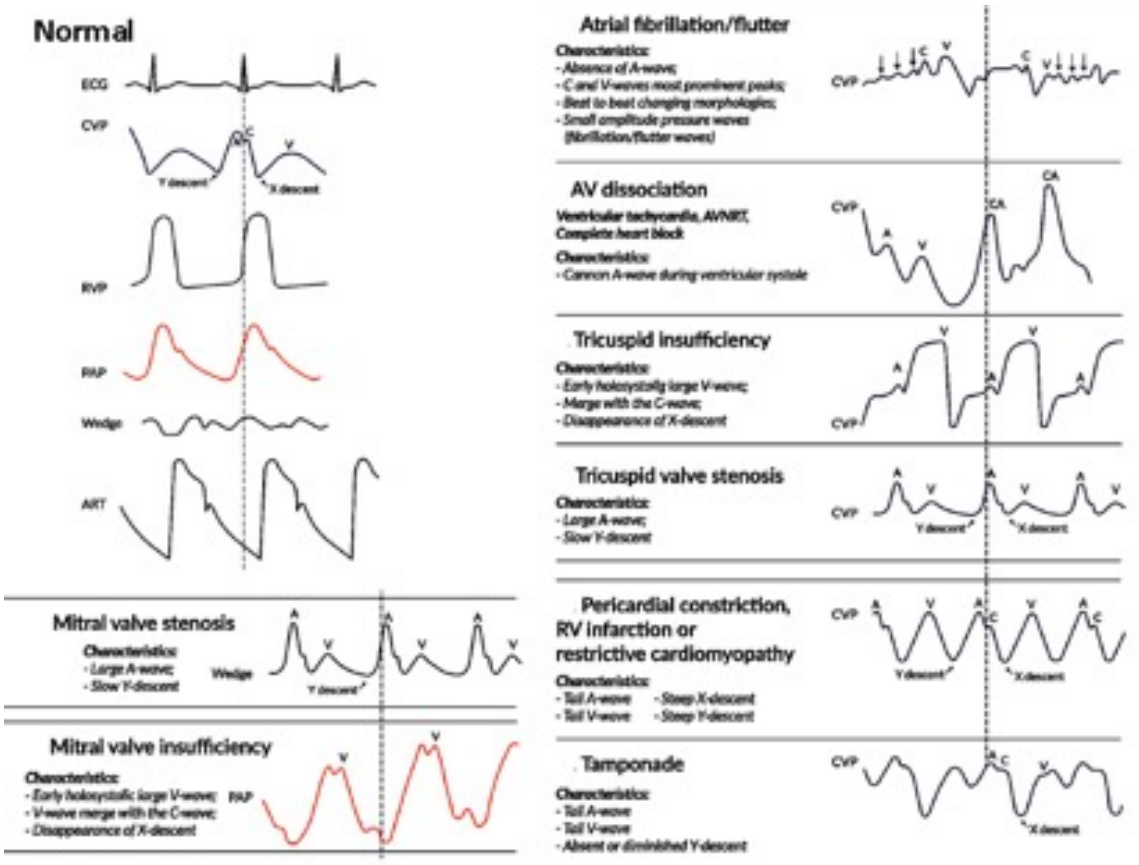
As described by **Bootsma et al,** RV contractile function is not synonymous with RV performance.[12] **Zhang et al** studied 518 mechanically ventilated patients of which 301 were categorized as normal RV contractile function, 164 with RV dysfunction but no evidence of venous congestion, and 53 had RV dysfunction with venous congestion.[58] The latter had longer ICU stay and more than 2x the mortality as the other two groups.[58] A CVP of 10 mmHg was a sensitive and specific cutoff to define congestion.[58] Hemodynamics are not stagnant, and assessment and monitoring should be continuous. In a study of 270 patients being evaluated for pulmonary hypertension undergoing exercise right heart catheterization, increases in RAP during exercise, even subtle increases (2-6 mmHg), were predictive was correlated with an increase in mean pulmonary artery pressure/cardiac output ratio (mPAP/CO), and predictive of mortality or lung transplantation.[59] In 76 patients with cardiogenic shock managed with mechanical circulatory support multivariate predictors of mortality, while the initial RAP was not a significant predictor, the changes from initial to final RAP was a predictor of mortality.[37]

CVP Waveform

The central venous or right atrial pressure monitoring yields more information than a pressure. The normal waveform results from atrial contraction, relaxation, pressures and flows, and reflects the functions of the right atrium, ventricle, valves, pericardial space, and rhythm (Figure 1).[34,60] The 'a' wave is the result of atrial contraction and pressure buildup to force blood into the RV. The 'c' wave occurs during isovolumic ventricular contraction and when the tricuspid valve is bulging into the RA and at time while blood is entering the RA from cava and hepatic veins. The 'v' wave occurs, during ventricular systole, when the atrial relaxation is slowing or finished and while blood is entering the RA from cava and hepatic vein. The 'x' decent follows atrial contraction, represents atrial relaxation and a reduction in pressure in systole allowing blood to enter. After the 'v' wave when the atrial pressure increased and the ventricle has relaxed, the tricuspid valve opens reducing atrial pressure represented by the 'y' descent. As blood flows down pressure gradients both the pressure and appearance of the RAP waveform will be impacted on.

Multiple diagnoses can be made based on the CVP and the CVP waveform.[34,35,60] (Figure 1). When the right ventricle fails, right atrial pressure and the waveform changes including an overall increase in amplitude.[54,61] As RV dysfunction will lead to venous congestion, correlation between RA, RV pressure and Doppler hepatic, renal, portal and femoral velocities have been reported (Figure 2).[77] This is important because abnormal splanchnic congestion measured using portal venous velocity is an independent predictor of worse outcome after cardiac surgery.[62,63] Therefore, earlier detection of abnormal RV dysfunction could initiate corrective therapy in order to avoid splanchnic congestion which precede multisystem organ dysfunction.[64]

Figure 1: Normal and abnormal right atrial pressure (RAP) hemodynamic waveforms



From Reference 34: Bootsma IT, Boerma EC, de Lange F, Scheeren TWL. The contemporary pulmonary artery catheter. Part 1: placement and waveform analysis. J Clin Monit Comput. 2022 Feb;36(1):5-15. doi: 10.1007/s10877-021-00662-8. Epub 2021 Feb 10. PMID: 33564995; PMCID: PMC8894225.

Figure 2: RV pressure and Doppler hepatic, renal, portal and femoral velocities

Invasive hemodynamic monitoring was able to predict the need for an RVAD in 115 patients after LVAD placement.[65] A higher RAP (9.5 vs 5.9; p = 0.001) and higher RAP/PCWP (0.49 vs 0.29; p < 0.001) were predictive of RVAD placement.[65] The benefits of monitoring the actual RAP waveform revealed that the 'y' descent was equal to or relatively greater depth than the 'x' descent, which is consistent with RV dilation and reduced compliance noted during atrial contraction, was a strong predictor of RVAD and added to the other predictors[65] (Figure 4).

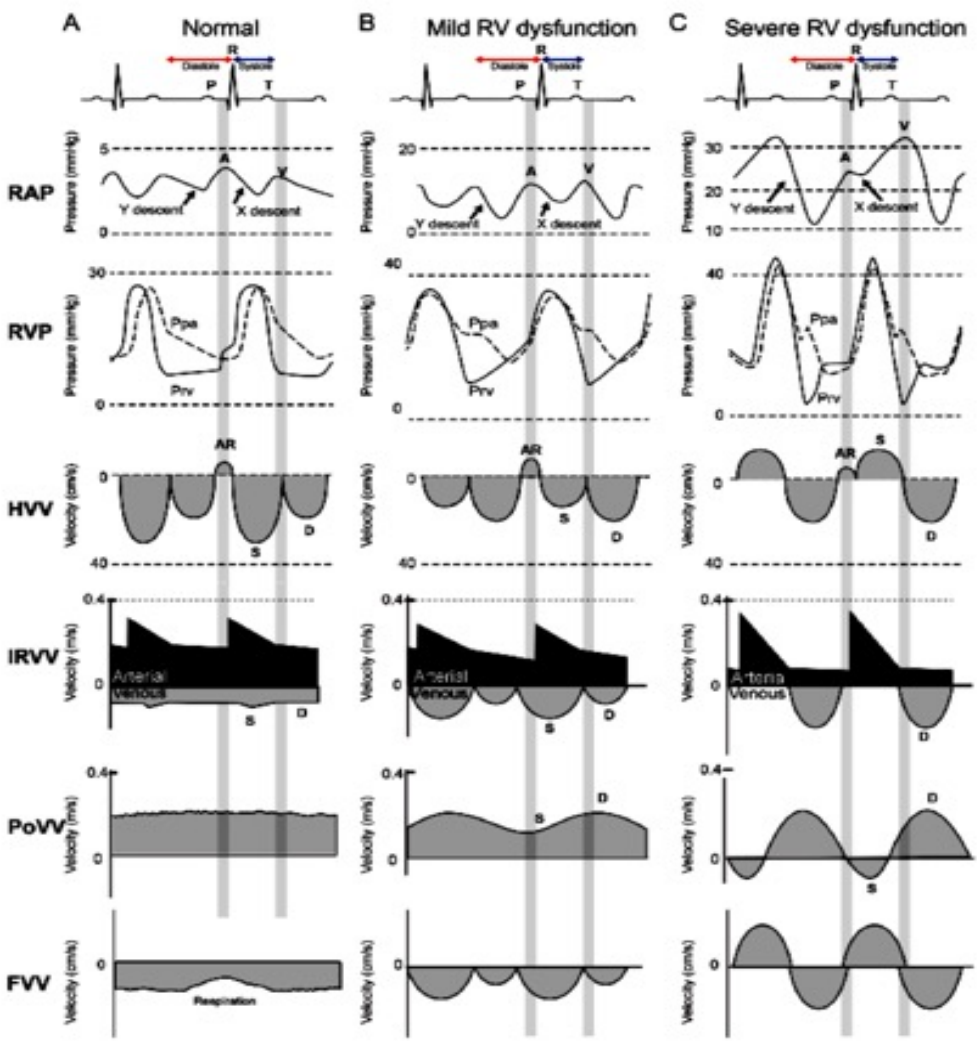


Figure 2 Figure 10.36 Doppler vein flow evaluation. Correlation between RAP waveform, Prv waveform, HVV, IRVV, PoVV, and FVV with progressive RV dysfunction and venous congestion. The typical patterns appear for (A) normal, (B) mild, and (C) severe RV dysfunction. Abbreviations: AR, atrial reversal HVV; D, diastolic velocity; FVV, femoral venous velocity; HVV, hepatic venous velocity; IRVV, interlobar renal venous velocity; Ppa, pulmonary artery pressure; Prv or RVP, right ventricular pressure; PoVV, portal venous velocity; RAP, right atrial pressure; RV, right ventricular; S, systolic velocity. Adapted from Couture EJ, Gronlykke L, Denault AY. New developments in the understanding of right ventricular function in acute care. Curr Opin Crit Care 2022; 28: 331-9. Doi: 10.1097/MCC.0000000000000946

In a study of 51 patients with or without RV ischemia, an increase in RAP amplitude and the development of an 'M' or 'W' pattern was predictive (> 90%) of RV ischemia and dysfunction, both associated with increased morbidity and mortality, and the former associated with greater right atrial pressures and a lower cardiac index[54] (Figure 3). The presence of the waveform difference was more predictive or RV ischemia than other hemodynamic measures.[54]

Figure 3: Right atrial and ventricular waveforms in patients with right ventricular ischemia and dysfunction. The figure to the left displays an 'W' pattern, while the one on the right displays an 'M' pattern.

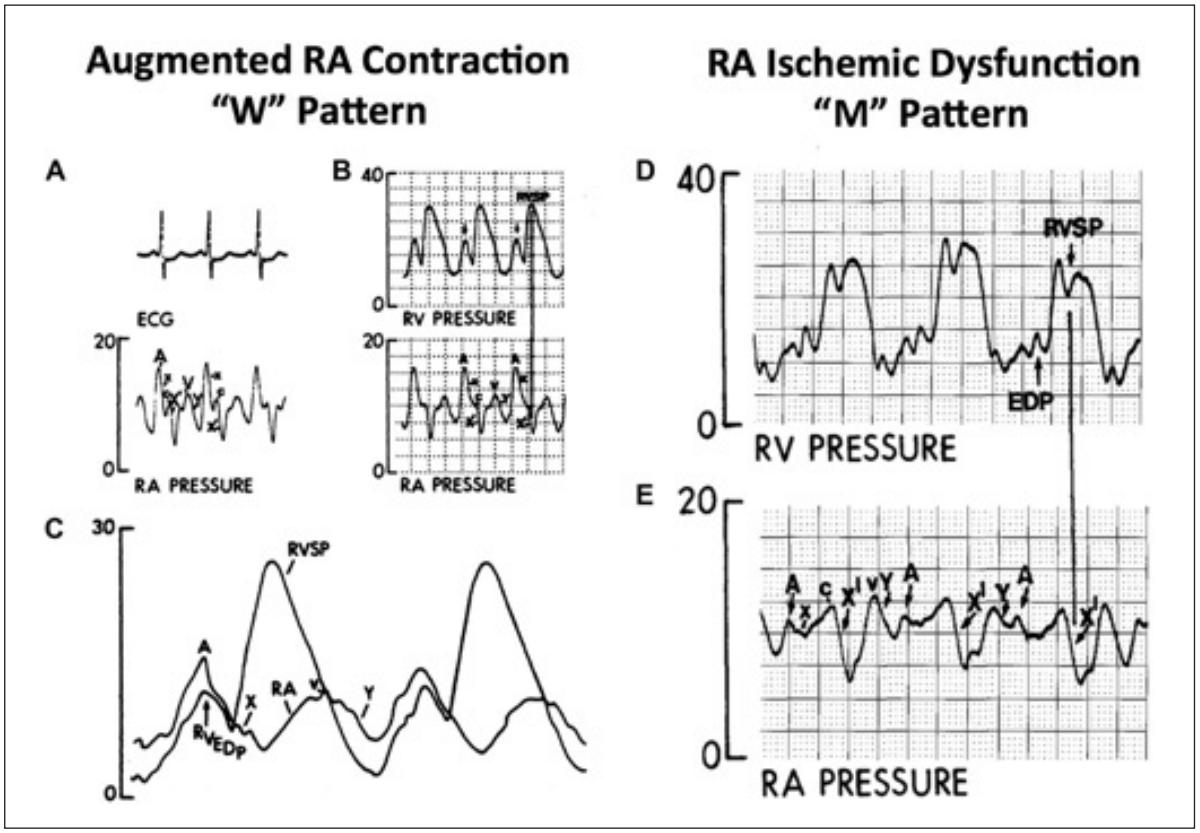


Image from: Goldstein JA. Hemodynamic Complications of Right Ventricular Infarction: Role of the Right Atrium. JACC Case Rep. 2021 Aug 4;3(9):1174-1176. doi: 10.1016/j jaccas.2021.02.045. PMID: 34401753; PMCID: PMC8353557.

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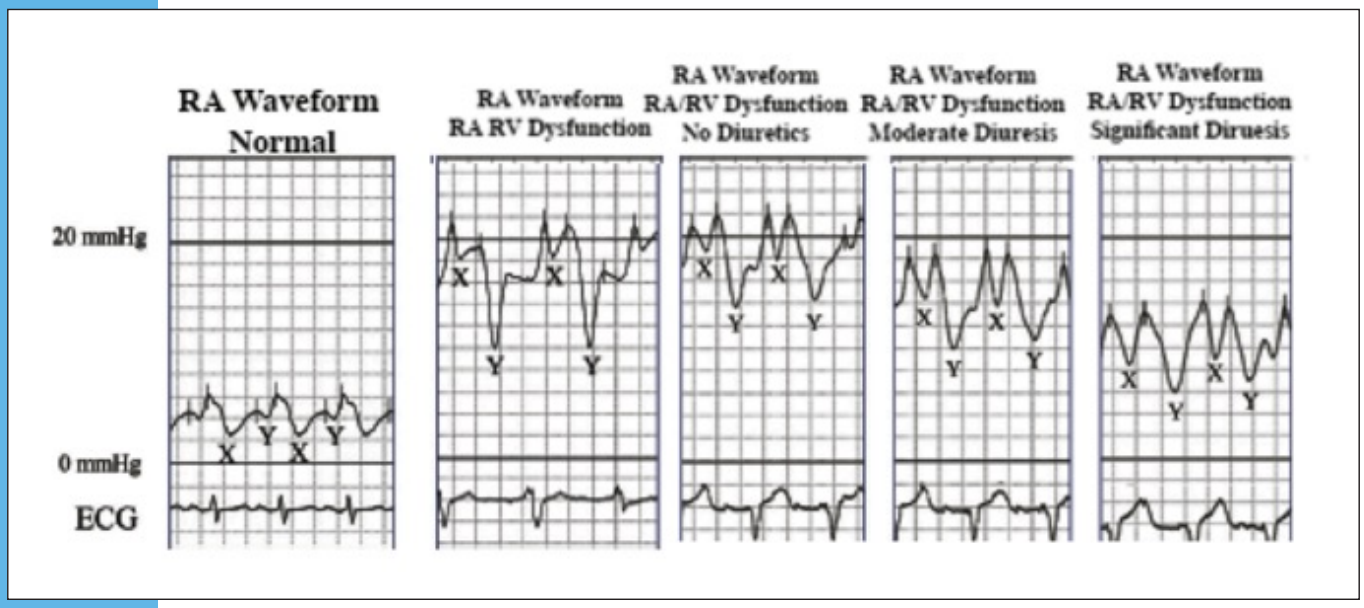
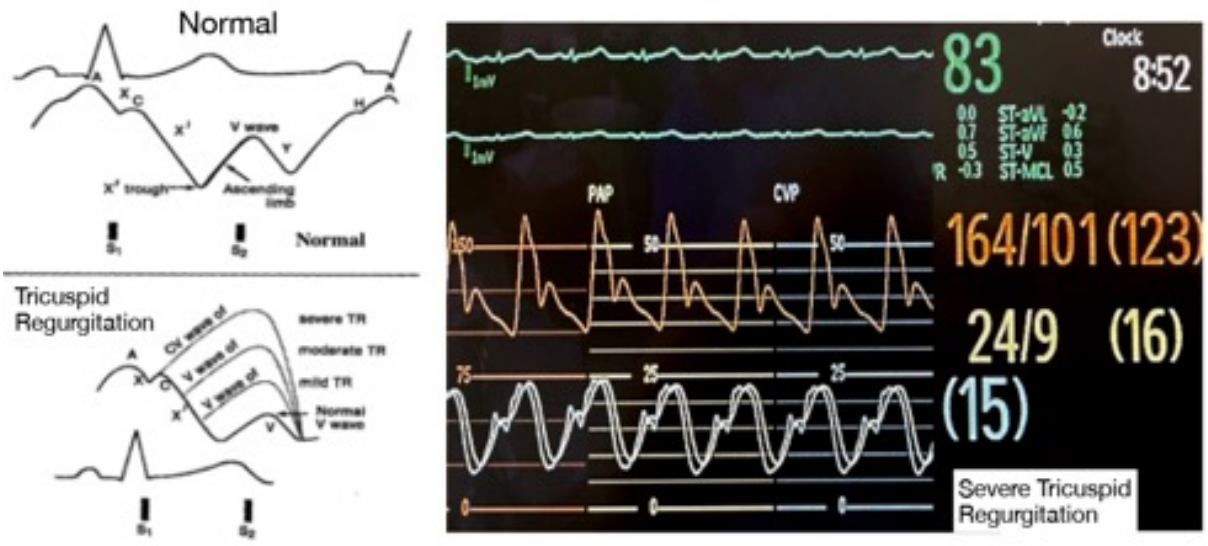
Figure 4: Relative depths of the 'x' and 'y' descents from right atrial (RA) waveforms with right ventricular dysfunction before and after diuresis. Note the change in 'x' descent before and after therapy.



From reference 65: Samura T, Yoshioka D, Asanoi H, Toda K, Miyagawa S, Yoshikawa Y, Hata H, Kainuma S, Kawamura T, Kawamura A, Sakata Y, Sawa Y. Right Atrial Pressure Waveform Predicts Right Ventricular Failure After Left Ventricular Assist Device Implantation. Ann Thorac Surg. 2019 Nov;108(5):1361-1368. doi: 10.1016/j.athoracsur.2019.04.050. Epub 2019 Jun 5. PMID: 31175868.

Figure 5: Tricuspid regurgitation seen on the right atrial pressure waveform. The normal example seen in the top left is compared to the progressively increasing regurgitation seen in the bottom left. Severe or torrential regurgitation is noted by the ventricularization in the right sided figure.



Elevated CVP or RAP occurs with pericardial pathologies.[66,67,68] With a pericardial effusion, venous congestion and hypertension is more than 90% sensitive of tamponade.[62] In addition, and especially relevant in cases of low-pressure tamponade, the 'x' and especially the 'y' descents are blunted reflecting the relatively greater pericardial pressure and impairment in filling.[66,67,68] (Figure 1). By contrast, while the 'a' and 'v' waves are increased in the presence of constrictive pericarditis, the 'x' and 'y' descents appear relatively steep.[67,68]

While monitoring for specific waveforms may be a highly skilled task, any changes in waveform and/or amplitude should alert the caregiver as to a hemodynamic change in right heart function/performance.

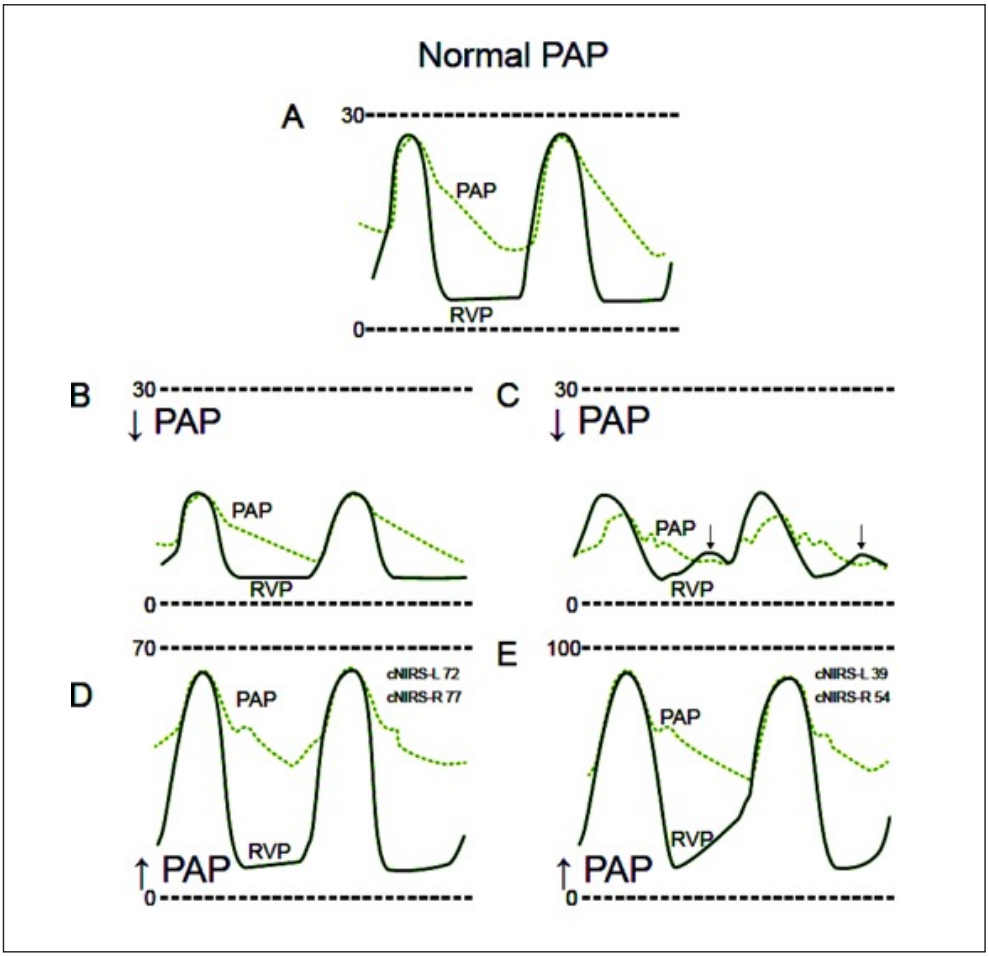
Right Ventricular Waveform Analysis

A pulmonary artery catheter (PAC) is often interchanged with right heart catheter however most high-volume centers using PACs in cardiac surgery completely ignore the RV pressure waveform except briefly during catheter insertion. Our center has been monitoring RV pressure waveform since 2002[69] initially for the diagnosis of RV outflow tract obstruction (RVOTO)[70,71] which can lead to suicide RV.[72,73,74] later for the diagnosis of RV diastolic dysfunction[75,76] and more recently for RV systolic function.[77,78,79] A detailed description of how to use it has been published.[69]

When using transesophageal echocardiography (TEE), would reporting only the size and velocities of the pulmonary artery and right atrium be an adequate way to assess right-sided cardiac function? Similarly, we fail to see the benefit of using a PAC limited only to pulmonary artery pressure (PAP) and right atrial pressure (RAP) monitoring without continuous RV pressure monitoring because the RV function is such a critical element in perioperative management and outcome. In our opinion, using a PAC without RV pressure monitoring is a missed opportunity in tracking RV critical systolic and diastolic in addition to RVOTO which has been reported prospectively to be present in up to one third of cardiac surgical patients.[70] given the widespread use of inotropes in that population.

The role of continuous RV pressure monitoring is illustrated in Figure 6[80] As shown in Figure 6A, if the PAP drops in an unstable patient, it can be secondary to hypovolemic or distributive shock (Figure 6B). In such a case, the systolic change in pressure over time (dP/dt) and the diastolic portion of the RV pressure waveform will remain normal. However, PAP reduction can also occur in RV failure, but in such a case the RV waveform will appear abnormal instantaneously: the change in systolic RV dP/dt will be prolonged and the diastolic pressure will gradually increase. There will be a diastolic equalization of the RV and PAP, a square-root sign and, in the worse cases, the RV diastolic pressure might raise above the diastolic PAP, leading to a diastolic opening of the pulmonic valve (Figure 6C; arrows), which has been described in severe RV heart failure (Figure 6C).[61] In the first case, volume, blood or vasopressors would be indicated to normalize cardiac output. In the presence of RV failure and limitation, volume and transfusion would contribute at one point to deteriorate right heart function. The use of a PAC with RV pressure waveform monitoring greatly simplifies the distinction between those two critical conditions and their treatment. The use of the pulmonary pulsatility index (PAPI) has been proposed to suspect RV dysfunction.[81] As RV function deteriorates, the PAP pulse pressure decreases and the RAP increase. However, PAPI has been shown to be unreliable in the presence of RVOTO.[82] which is present in more than 40% of patents after separation from cardiopulmonary bypass.[70] Again, the use of RV pressure monitoring as an index of RV dysfunction or RV failure is far superior to identify abnormal RV function compared to the use of using of PAP and RAP.

Figure 6: Continuous right ventricular pressure (RVP) and pulmonary artery pressure (PAP) monitoring



RV failure can be defined as abnormal ventricular arterial (VA) coupling.[62] How can we tell if RV is coupled or uncoupled with its afterload when PH appears? Elevated PAP can occur when the RV is recovering from an acute insult and systolic function is improving. In such a situation, the RV systolic function will be adapted to elevated PAP and the diastolic function will often be normal, with normal tissue oxygenation (Figure 6D). However, elevated PAP can occur with abnormal VA coupling. In such a case, the diastolic component of the RV waveform will be abnormal, showing a rapidly elevated pressure during filling and tissue oxygenation compromise (Figure 6E). In other words, the use of continuous RV and PA pressure monitoring allows instantaneous detection of RV VA coupling. For instance, the use of RV pressure monitoring can be used to demonstrate that the efficacy of combined inhaled prostacyclin and milrinone which do improve both RV contractility and RV afterload. Responders to combined inhaled agents will be demonstrate an increase in heart rate, mean arterial pressure and both RV and LV dp/dt. As the severity of PH improve there will be lowering of the mean PAP, RVEDP and in the RV diastolic gradient. Interestingly an increase in the PA to RV systolic gradient will be observed as RV contractility increase.[78]

The value of hemodynamic assessment cannot be overstated. Without dismissing the value of perioperative transthoracic and transesophageal echocardiography, caregivers must recognize its limitations in estimating pressures and flows, assessing geometry, volume and contractile performance, and the need for an expertly trained sonographer/clinician to assess data and frequently adjust the imaging probe angle and position. By contrast, the presence of invasive monitoring permits continuous measurement and assessment of the full breath of hemodynamic data for all levels of caregivers to interpret and use to assess and guide therapy. If the criticism of invasive monitoring is the understanding of the numbers then it is our job to teach it better. Although waveform analysis is an additional skill, at a minimum caregivers should be able to recognize any changes in appearance and or amplitude.

It is only a matter of time before artificial intelligence will improve utilization of both echocardiography based and pulmonary artery catheter-based data for all caregivers.[83,84] It is reasonable to assume that the more educated caregivers are to the hemodynamic data presented, the better they will be at assessing and managing patients, ultimately hoping to improve outcome.[85]

1 Torrent-Guasp F, Kocica MJ, Corno AF et al: Towards new understanding of the heart structure and function. Eur J Cardiothorac Surg 2005;27:191-201.

2 Kocica MJ, Corno AF, Carreras-Costa F et al: The helical ventricular myocardial band: global, three-dimensional, functional architecture of the ventricular myocardium, European Journal of Cardio-Thoracic Surgery, Volume 2006;29:S21–S40. https://doi.org/10.1016/j.ejcts.2006.03.011

3 Damiano RJ, Jr, La Follette P Jr, Cox JL, Lowe JE, Santamore WP: Significant left ventricular contribution to right ventricular systolic function. Am J Physiol 1991;261:H1514–H1524.

4 Fenely MP, Olsen CO, Glower DD, Rankin JS: Effect of acutely increased right ventricular afterload on work output from the left ventricle in conscious dogs. Systolic ventricular interaction. Circ Res 1989;65:135-145.

5 Goldstein JA, Tweddell JS, Barzilai B, Yagi Y, Jaffe AS, Cox JL. Importance of left ventricular function and systolic ventricular interaction to right ventricular performance during acute right heart ischemia. J Am Coll Cardiol. 1992 Mar 1;19(3):704-11. doi: 10.1016/s0735-1097(10)80296-7. PMID: 1538031.

6. Hoffman D, Sisto D, Frater RWM, Nikilic SD: Left-to-right ventricular interaction with a noncontracting right ventricle. J Thorac Cardiovasc Surg 1994;107:1496-1502.

7 Danton MH, Byrne JG, Flores KQ, Laurence RG, Cohn LH, Aklog L: Modified Glenn connection for acutely ischemic right ventricular failure reverses secondary left ventricular dysfunction. J Thorac Cardiovasc Surg 2001;122:80-91.

8 Lin SS, Reynertson SI, Louie EK, Levitsky S : Right ventricular volume overload results in depression of left ventricular ejection fraction. Implications for the surgical management of tricuspid valve disease. Circulation 1994;90:II209-213.

9 Chen L, Hong L, Ma A, Chen Y, Xiao Y, Jiang F, Huang R, Zhang C, Bu X, Ge Y, Zhou J: Intraoperative venous congestion rather than hypotension is associated with acute adverse kidney events after cardiac surgery: a retrospective cohort study British Journal of Anaesthesia 2022;128(5):785e795 doi: 10.1016/j.bja.2022.01.032

10 Ross EA. Congestive renal failure: the pathophysiology and treatment of renal venous hypertension. J Card Fail. 2012 Dec;18(12):930-8. doi: 10.1016/j.cardfail.2012.10.010. Epub 2012 Oct 27. PMID: 23207082.

11 Zochios V, Protopapas AD, Parhar K: Markers of right ventricular dysfunction in adult cardiac surgical patients. J Cardiothorac Vasc Anesth 2017;31:1570-1574.

12 Bootsma IT, Scheeren TWL, de Lange F, Jainandunsing JS, Boerma EC. The Reduction in Right Ventricular Longitudinal Contraction Parameters Is Not Accompanied by a Reduction in General Right Ventricular Performance During Aortic Valve Replacement: An Explorative Study. J Cardiothorac Vasc Anesth. 2020 Aug;34(8):2140-2147. doi: 10.1053/j.jvca.2020.01.012. Epub 2020 Jan 15. PMID: 32139346.

13 Haddad F, Couture P, Tousignant C, Denault AY. The right ventricle in cardiac surgery, a perioperative perspective: I. Anatomy, physiology, and assessment. Anesth Analg. 2009 Feb;108(2):407-21. doi: 10.1213/ane.0b013e31818f8623. PMID: 19151264.

14 Haddad F, Couture P, Tousignant C, Denault AY. The right ventricle in cardiac surgery, a perioperative perspective: II. Pathophysiology, clinical importance, and management. Anesth Analg. 2009 Feb;108(2):422-33. doi: 10.1213/ane.0b013e31818d8b92. PMID: 19151265.

15 Haddad, François MD*†; Couture, Pierre MD*; Tousignant, Claude MD‡; Denault, André Y. MD*. The Right Ventricle in Cardiac Surgery, a Perioperative Perspective: II. Pathophysiology, Clinical Importance, and Management. Anesthesia & Analgesia 108(2):p 422-433, February 2009. | DOI: 10.1213/ane.0b013e31818d8b92

16 Kagan A: Dynamic responses of the right ventricle following extensive damage by cauterization. Circulation 19525:816-823.

17 Sawatani S, Mandell C, Kusaba E: Ventricular performance following ablation and prosthetic replacement of right ventricular myocardium. Trans Am Soc Artif Intern Organs. 1974;20:629-636.

18 Starr I, Jeffers W, Meade Jr RH: The absence of conspicuous increments of venous pressure after severe damage to the right ventricle of the dog, with a discussion of the relation between clinical congestive failure and heart disease. Am Heart J 1953;26:291-301.

19 Fontan F, Baudet E: Surgical repair of tricuspid atresia. Thorax 1971;26:240-248.

20 Pundi KN, Johnson JN, Dearani JA et al: 40 year follow-up after the Fontan operation: Long-term outcomes of 1052 patients. J Am Coll Cardiol 2015;13:1700-1710.

21 Dennis M, Zannino D, du Plessis K et al: Clinical Outcomes in Adolescents and Adults After the Fontan Procedure. J Am Coll Cardiol. 2018;71:1009-1017.

22 Khairy P, Ferandes SM, Mayer Jr JE et al: Long-term survival, modes of death, and predictors of mortality in patients with Fontan Surgery. Circulation 2008;117:85-92.

23 Naeijie R, Brimioulle S, Dewachter L: Biomechanics of the right ventricle in health and disease (2013 Grover Conference Series). Pulm Circ 2014;4:395-406.

24 Vonk-Noordegraaf A, Chin KM, Haddad F et al: Pathophysiology of the right ventricle and of the pulmonary circulation in pulmonary hypertension: an update. Eur Respir J 2019;53:1801900 https://doi.org/10.1183/13993003.01900-2018

25 Vonk-Noordegraaf A, Westerhof BE, Westerhof N: The Relationship Between the Right Ventricle and its Load in Pulmonary Hypertension. J Am Coll Cardiol 2017;69:236-243.

26 Todaro MC, Carerj S, Trifiro MP, Consolo G, Khandheria B: Echocardiographic evaluation of right ventricular-arterial coupling in pulmonary hypertension. Am J Cardiovasc Dis. 2020;10;272-283. SSN:2160-200X/AJCD0116854

27 Bellofiore A, Chesler NC. Methods for measuring right ventricular function and hemodynamic coupling with the pulmonary vasculature. Ann Biomed Eng. 2013 Jul;41(7):1384-98. doi: 10.1007/s10439-013-0752-3. Epub 2013 Feb 20. PMID: 23423705; PMCID: PMC3679286.

28 Sanz J, Sánchez-Quintana D, Bossone E, Bogaard HJ, Naeije R. Anatomy, Function, and Dysfunction of the Right Ventricle: JACC State-of-the-Art Review. J Am Coll Cardiol. 2019 Apr 2;73(12):1463-1482. doi: 10.1016/j.jacc.2018.12.076. PMID: 30922478.

29 Maslow AD, Regan MM, Schwartz C, Bert A, Singh A. Inotropes improve right heart function in patients undergoing aortic valve replacement for aortic stenosis. Anesth Analg. 2004 Apr;98(4):891-902. doi: 10.1213/01.ANE.0000107940.23783.33. PMID: 15041568.

30 Hameed A, Condliffe R, Swift AJ, Alabed S, Kiely DG, Charalampopoulos A. Assessment of Right Ventricular Function-a State of the Art. Curr Heart Fail Rep. 2023 Jun;20(3):194-207. doi: 10.1007/s11897-023-00600-6. Epub 2023 Jun 5. PMID: 37271771; PMCID: PMC10256637.

31 Ivan H W Yim, Ayisha M Khan-Kheil, Nigel E Drury, Hoong Sern Lim, A systematic review and physiology of pulmonary artery pulsatility index in left ventricular assist device therapy, Interdisciplinary CardioVascular and Thoracic Surgery, Volume 36, Issue 5, May 2023, ivad068,

32 Naeije R, Manes A. The right ventricle in pulmonary arterial hypertension. Eur Respir Rev. 2014 Dec;23(134):476-87. doi: 10.1183/09059180.00007414. PMID: 25445946; PMCID: PMC9487395.

33 Naeije R, Chin K: Differentiating precapillary from postcapillary pulmonary hypertension. Circulation 2019;140:712-714. DOI: 10.1161/CIRCULATIONAHA.119.040295

34 Bootsma IT, Boerma EC, de Lange F, Scheeren TWL. The contemporary pulmonary artery catheter. Part 1: placement and waveform analysis. J Clin Monit Comput. 2022 Feb;36(1):5-15. doi: 10.1007/s10877-021-00662-8. Epub 2021 Feb 10. PMID: 33564995; PMCID: PMC8894225.

35 Bootsma IT, Boerma EC, Scheeren TWL, de Lange F. The contemporary pulmonary artery catheter. Part 2: measurements, limitations, and clinical applications. J Clin Monit Comput. 2022 Feb;36(1):17-31. doi: 10.1007/s10877-021-00673-5. Epub 2021 Mar 1. PMID: 33646499; PMCID: PMC7917533.

36 Rong LQ, Kaushal M, Mauer E, Pryor KO, Kenfield M, Shore-Lesseron L, Gaudino MFL, Neuburger PJ. Two- or 3-Dimensional Echocardiography-Derived Cardiac Output Cannot Replace the Pulmonary Artery Catheter in Cardiac Surgery. J Cardiothorac Vasc Anesth. 2020 Oct;34(10):2691-2697. doi: 10.1053/j.jvca.2020.06.068. Epub 2020 Jun 24. PMID: 32693966.

37 Davila CD, Esposito M, Hirst CS, Morine K, Jorde L, Newman S, Paruchuri V, Whitehead E, Thayer KL, Kapur NK. Right Atrial Pressure Is Associated With Outcomes in Patient With Cardiogenic Shock Receiving Acute Mechanical Circulatory Support. Front Cardiovasc Med. 2021 Feb 11;8:563853. doi: 10.3389/fcvm.2021.563853. PMID: 33644126; PMCID: PMC7905221.

38 Ma TS, Bozkurt B, Paniagua D, Kar B, Ramasubbu K, Rothe CF. Central venous pressure and pulmonary capillary wedge pressure: fresh clinical perspectives from a new model of discordant and concordant heart failure. Tex Heart Inst J. 2011;38(6):627-38. PMID: 22199422; PMCID: PMC3233309.

39 Woodard JC, Chow E, Farrar DJ. Isolated ventricular systolic interaction during transient reductions in left ventricular pressure. Circ Res. 1992 May;70(5):944-51. doi: 10.1161/01.res.70.5.944. PMID: 1568303.

40 Klima UP, Lee J-Y, Guerrero JL, Laraia PJ, Levine RA, Vlahakes GJ: Determinants of maximal right ventricular function; Role of septal shift. J Thorac Cardiovasc Surg 2002;123:72-80.

41 Belenkie et al: Effects of aortic constriction during experimental acute right ventricular pressure loading. Circulation 1995;92:546-554.

42 Farber HW, Miller DP, Poms AD, Badesch DB, Frost AE, Muros-Le Rouzic E, Romero AJ, Benton WW, Elliott CG, McGoon MD, Benza RL. Five-Year outcomes of patients enrolled in the REVEAL Registry. Chest. 2015 Oct;148(4):1043-54. doi: 10.1378/chest.15-0300. PMID: 26066077.

43 Benza RL, Miller DP, Gomberg-Maitland M, Frantz RP, Foreman AJ, Coffey CS, Frost A, Barst RJ, Badesch DB, Elliott CG, Liou TG, McGoon MD. Predicting survival in pulmonary arterial hypertension: insights from the Registry to Evaluate Early and Long-Term Pulmonary Arterial Hypertension Disease Management (REVEAL). Circulation. 2010 Jul 13;122(2):164-72. doi: 10.1161/CIRCULATIONAHA.109.898122. Epub 2010 Jun 28. PMID: 20585012.

44 Vizza CD, Lang IM, Badagliacca R, Benza RL, Rosenkranz S, White RJ, Adir Y, Andreassen AK, Balasubramanian V, Bartolome S, Blanco I, Bourge RC, Carlsen J, Camacho REC, D'Alto M, Farber HW, Frantz RP, Ford HJ, Ghio S, Gomberg-Maitland M, Humbert M, Naeije R, Orfanos SE, Oudiz RJ, Perrone SV, Shlobin OA, Simon MA, Sitbon O, Torres F, Luc Vachiery J, Wang KY, Yacoub MH, Liu Y, Golden G, Matsubara H. Aggressive Afterload Lowering to Improve the Right Ventricle: A New Target for Medical Therapy in Pulmonary Arterial Hypertension? Am J Respir Crit Care Med. 2022 Apr 1;205(7):751-760. doi: 10.1164/rccm.202109-2079PP. PMID: 34905704; PMCID: PMC9836222.

45 Price LC, Wort SJ, Finney SJ, Marino PS, Brett SJ: Pulmonary vascular and right ventricular dysfunction in adult critical care: current and emerging options for management: a systematic literature review. Crit Care Med 2010;14:R169. http://ccforum.com/content/14/5/R169

46 Pi H, Kosanovich CM, Handen A et al: Outcomes of pulmonary arterial hypertension are improved in a specialty care center. Chest 2020;158:330-340. DOI: https://doi.org/10.1016/j.chest.2020.01.046

47 Ramakrishna G, Sprung J, Ravi BS, Chandrasekaran K, McGoon MD: Impact of pulmonary hypertension on the outcomes of noncardiac surgery. J Am Coll Cardiol 2005;46:1691-1699. doi:10.1016/j.jacc.2005.02.055

48 Lourenco AP, Fontoura D, Henriques-Coelho T, Leit-Moreira AF: Current pathophysiological concepts and management of pulmonary hypertension. Intern J Cardiol 2012;155;350-361. doi:10.1016/j.ijcard.2011.05.066

49 Price LC, Martinez G, Brame A et al: Perioperative management of patients with pulmonary hypertension undergoing non-cardiothoracic, non-obstetric surgery: a systematic review and expert consensus statement. Brit J Anaesth 2021;126;774-790. doi: 10.1016/j.bja.2021.01.005

50 Kochar A, Zheng Y, van Diepen S, Mehta RH, Westerhout CM, Mazer DC, Duncan AI, Whitlock R, Lopes RD, Argenziano M, de Varennes B, Alexander JH, Goodman SG, Fremes S. Predictors and associated clinical outcomes of low cardiac output syndrome following cardiac surgery: insights from the LEVO-CTS trial. Eur Heart J Acute Cardiovasc Care. 2022 Nov 30;11(11):818-825. doi: 10.1093/ehjacc/zuac114. PMID: 36156131.

51 Rao V, Ivanov J, Weisel RD, Ikonomidis JS, Christakis GT, David TE. Predictors of low cardiac output syndrome after coronary artery bypass. J Thorac Cardiovasc Surg. 1996 Jul;112(1):38-51. doi: 10.1016/s0022-5223(96)70176-9. PMID: 8691884.

52 Chemla D, Castelain V, Zhu K, Papelier Y, Creuzé N, Hoette S, Parent F, Simonneau G, Humbert M, Herve P. Estimating right ventricular stroke work and the pulsatile work fraction in pulmonary hypertension. Chest. 2013 May;143(5):1343-1350. doi: 10.1378/chest.12-1880. PMID: 23715528.

53 Task Force for Diagnosis and Treatment of Pulmonary Hypertension of European Society of Cardiology (ESC); European Respiratory Society (ERS); International Society of Heart and Lung Transplantation (ISHLT); Galiè N, Hoeper MM, Humbert M, Torbicki A, Vachiery JL, Barbera JA, Beghetti M, Corris P, Gaine S, Gibbs JS, Gomez-Sanchez MA, Jondeau G, Klepetko W, Opitz C, Peacock A, Rubin L, Zellweger M, Simonneau G. Guidelines for the diagnosis and treatment of

pulmonary hypertension. Eur Respir J. 2009 Dec;34(6):1219-63. doi: 10.1183/09031936.00139009. Epub 2009 Sep 12. PMID: 19749199.

54 Cohen A, Guyon P, Johnson N, Chauvel C, Logeart D, Costagliola D, Valty J. Hemodynamic criteria for diagnosis of right ventricular ischemia associated with inferior wall left ventricular acute myocardial infarction. Am J Cardiol. 1995 Aug 1;76(4):220-5. doi: 10.1016/s0002-9149(99)80069-1. PMID: 7618612.

55 Chen H, Zhu Z, Zhao C, Guo Y, Chen D, Wei Y, Jin J. Central venous pressure measurement is associated with improved outcomes in septic patients: an analysis of the MIMIC-III database. Crit Care. 2020 Jul 14;24(1):433. doi: 10.1186/s13054-020-03109-9. PMID: 32665010; PMCID: PMC7358999.

56 Chen H, Gong SR, Shang XL, Li J, Yu RG. The magnitude, but not the duration of elevated central venous pressure is associated with mortality in sepsis patients: An analysis of the MIMIC-IV database. PLoS One. 2023 Feb 8;18(2):e0281549. doi: 10.1371/journal.pone.0281549. PMID: 36753503; PMCID: PMC9907836.

57 Huang A, Liao L, Pan L, Pinhu L. Association Between the Central Venous Pressure and All-Cause Mortality in Critically Ill Patients with Acute Kidney Injury. Int J Gen Med. 2021 Nov 10;14:8019-8027. doi: 10.2147/IJGM.S331686. PMID: 34785944; PMCID: PMC8590983.

58 Zhang H, Lian H, Zhang Q, Zhao H, Wang X. Can central venous pressure help identify acute right ventricular dysfunction in mechanically ventilated critically ill patients? Ann Intensive Care. 2024 Jul 20;14(1):114. doi: 10.1186/s13613-024-01352-9. PMID: 39031301; PMCID: PMC11264666.

59 Lichtblau M, Bader PR, Saxer S, Berlier C, Schwarz EI, Hasler ED, Furian M, Grünig E, Bloch KE, Ulrich S. Right Atrial Pressure During Exercise Predicts Survival in Patients With Pulmonary Hypertension. J Am Heart Assoc. 2020 Nov 17;9(22):e018123. doi: 10.1161/JAHA.120.018123. Epub 2020 Nov 4. PMID: 33146048; PMCID: PMC7763735.

60 Magder S. Right Atrial Pressure in the Critically Ill: How to Measure, What Is the Value, What Are the Limitations? Chest. 2017 Apr;151(4):908-916. doi: 10.1016/j.chest.2016.10.026. Epub 2016 Nov 1. PMID: 27815151.

61 Doyle T, Troup PJ, Wann LS. Mid-diastolic opening of the pulmonary valve after right ventricular infarction. J Am Coll Cardiol. 1985 Feb;5(2 Pt 1):366-8. doi: 10.1016/s0735-1097(85)80061-9. PMID: 3968320.

62 Eljaiek R, Cavayas YA, Rodrigue E, Desjardins G, Lamarche Y, Toupin F, Denault AY, Beaubien-Souligny W. High postoperative portal venous flow pulsatility indicates right ventricular dysfunction and predicts complications in cardiac surgery patients. Br J Anaesth. 2019 Feb;122(2):206-214. doi: 10.1016/j.bja.2018.09.028. Epub 2018 Nov 28. PMID: 30686306.

63 Denault A, Couture EJ, De Medicis É, Shim JK, Mazzeffi M, Henderson RA, Langevin S, Dhawan R, Michaud M, Guensch DP, Berger D, Erb JM, Gebhard CE, Royse C, Levy D, Lamarche Y, Dagenais F, Deschamps A, Desjardins G, Beaubien-Souligny W. Perioperative Doppler ultrasound assessment of portal vein flow pulsatility in high-risk cardiac surgery patients: a multicentre prospective cohort study. Br J Anaesth. 2022 Nov;129(5):659-669. doi: 10.1016/j.bja.2022.07.053. Epub 2022 Sep 30. PMID: 36184294.

64 Grignola JC, Domingo E. Acute Right Ventricular Dysfunction in Intensive Care Unit. Biomed Res Int. 2017;2017:8217105. doi: 10.1155/2017/8217105. Epub 2017 Oct 19. PMID: 29201914; PMCID: PMC5671685.

65 Samura T, Yoshioka D, Asanoi H, Toda K, Miyagawa S, Yoshikawa Y, Hata H, Kainuma S, Kawamura T, Kawamura A, Sakata Y, Sawa Y. Right Atrial Pressure Waveform Predicts Right Ventricular Failure After Left Ventricular Assist Device Implantation. Ann Thorac Surg. 2019 Nov;108(5):1361-1368. doi: 10.1016/j.athoracsur.2019.04.050. Epub 2019 Jun 5. PMID: 31175868.

66 Carmona P, Mateo E, Casanovas I, Peña JJ, Llagunes J, Aguar F, De Andrés J, Errando C. Management of cardiac tamponade after cardiac surgery. J Cardiothorac Vasc Anesth. 2012 Apr;26(2):302-11. doi: 10.1053/j.jvca.2011.06.007. Epub 2011 Aug 25. Erratum in: J Cardiothorac Vasc Anesth. 2012 Aug;26(4):758. PMID: 21868250.

67 Shabetai R, Fowler NO, Guntheroth WG. The hemodynamics of cardiac tamponade and constrictive pericarditis. Am J Cardiol. 1970 Nov;26(5):480-9. doi: 10.1016/0002-9149(70)90706-x. PMID: 5478837.

68 Klein AL, Wang TKM, Cremer PC, Abbate A, Adler Y, Asher C, Brucato A, Chetrit M, Hoit B, Jellis CL, Kwon DH, LeWinter M, Lin D, Luis SA, Mardigyan V, Oh JK, Ordovas KG, Rodriugez ER, Schenone AL, Tan CD, Weber B, Imazio M. Pericardial Diseases: International Position Statement on New Concepts and Advances in Multimodality Cardiac Imaging. JACC Cardiovasc Imaging. 2024 Aug;17(8):937-988. doi: 10.1016/j.jcmg.2024.04.010. PMID: 39111992.

69 Raymond M, Grønlykke L, Couture EJ, Desjardins G, Cogan J, Cloutier J, Lamarche Y, L'Allier PL, Ravn HB, Couture P, Deschamps A, Chamberland ME, Ayoub C, Lebon JS, Julien M, Taillefer J, Rochon A, Denault AY. Perioperative Right Ventricular Pressure Monitoring in Cardiac Surgery. J Cardiothorac Vasc Anesth. 2019 Apr;33(4):1090-1104. doi: 10.1053/j.jvca.2018.08.198. Epub 2018 Aug 25. PMID: 30269893.

70 Couture EJ, Calderone A, Zeng YH, Jarry S, Saade E, Hammoud A, Elmi-Sarabi M, Beaubien-Souligny W, Denault A. Instantaneous Right Ventricular to Pulmonary Artery Systolic Pressure Difference in Cardiac Surgery: A Retrospective and Prospective Cohort Study. Can J Cardiol. 2024 Aug 8:S0828-282X(24)00849-3. doi: 10.1016/j.cjca.2024.08.262. Epub ahead of print. PMID: 39127257.

71 Denault AY, Chaput M, Couture P, Hebert Y, Haddad F, Tardif JC. Dynamic right ventricular outflow tract obstruction in cardiac surgery. J Thorac Cardiovasc Surg. 2006;132(1):43-9.

72 Singhal A, Kumar S, Kapoor A. Sudden iatrogenic suicidal right ventricle. Indian Heart J. 2015 Jul-Aug;67(4):406-8. doi: 10.1016/j.ihj.2015.06.043. Epub 2015 Jul 23. PMID: 26304582; PMCID: PMC4561786.

73 Gangahanumaiah S, Scarr BC, Buckland MR, Pilcher DV, Paraskeva MA, McGiffin DC. Suicide right ventricle after lung transplantation for pulmonary vascular disease. J Card Surg. 2018;33(7):412-5.

74 Zeng YH, Calderone A, Rousseau-Saine N, Elmi-Sarabi M, Jarry S, Couture ÉJ, Aldred MP, Dorval JF, Lamarche Y, Miles LF, Beaubien-Souligny W, Denault AY. Right Ventricular Outflow Tract Obstruction in Adults: A Systematic Review and Meta-analysis. CJC Open. 2021 Apr 9;3(9):1153-1168. doi: 10.1016/j.cjco.2021.03.011. PMID: 34746729; PMCID: PMC8551422.

75 Grønlykke L, Couture EJ, Haddad F, Amsallem M, Ravn HB, Raymond M, Beaubien-Souligny W, Demers P, Rochon A, Sarabi ME, Lamarche Y, Desjardins G, Denault AY. Preliminary Experience Using Diastolic Right Ventricular Pressure Gradient Monitoring in Cardiac Surgery. J Cardiothorac Vasc Anesth. 2020 Aug;34(8):2116-2125. doi: 10.1053/j.jvca.2019.12.042. Epub 2020 Jan 8. PMID: 32037274.

76 Denault A, Couture EJ, Perry T, Saade E, Calderone A, Zeng YH, Scherb D, Moses K, Potes C, Hammoud A, Beaubien-Souligny W, Elmi-Sarabi M, Grønlykke L, Lamarche Y, Lebon JS, Rousseau-Saine N, Desjardins G, Rochon A. Continuous Right Ventricular Pressure Monitoring in Cardiac Surgery. J Cardiothorac Vasc Anesth. 2024 Aug;38(8):1673-1682. doi: 10.1053/j.jvca.2024.04.025. Epub 2024 Apr 22. PMID: 38862285.

77 Calderone A, Jarry S, Couture EJ, Brassard P, Beaubien-Souligny W, Momeni M, Liszkowski M, Lamarche Y, Shaaban-Ali M, Matta B, Rochon A, Lebon JS, Ayoub C, Martins MR, Courbe A, Deschamps A, Denault AY. Early Detection and Correction of Cerebral Desaturation With Noninvasive Oxy-Hemoglobin, Deoxy-Hemoglobin, and Total Hemoglobin in Cardiac Surgery: A Case Series. Anesth Analg. 2022 Dec 1;135(6):1304-1314. doi: 10.1213/ANE.0000000000006155. Epub 2022 Nov 16. PMID: 36097147.

78 Elmi-Sarabi M, Couture E, Jarry S, Saade E, Calderone A, Potes C, Denault A. Inhaled Epoprostenol and Milrinone Effect on Right Ventricular Pressure Waveform Monitoring. Can J Cardiol. 2023 Apr;39(4):474-482. doi: 10.1016/j.cjca.2022.12.007. Epub 2022 Dec 14. PMID: 36528279.

79 Elmi-Sarabi M, Jarry S, Couture EJ, Haddad F, Cogan J, Sweatt AJ, Rousseau-Saine N, Beaubien-Souligny W, Fortier A, Denault AY. Pulmonary Vasodilator Response of Combined Inhaled Epoprostenol and Inhaled Milrinone in Cardiac Surgical Patients. Anesth Analg. 2023 Feb 1;136(2):282-294. doi: 10.1213/ANE.0000000000006192. Epub 2022 Sep 19. PMID: 36121254.

80 Denault A, Canevet M, Couture EJ. Pro: We Should Use a Pulmonary Artery Catheter with Right Ventricular Pressure Waveforms in Cardiac Surgical Patients. J Cardiothorac Vasc Anesth. 2023 Apr;37(4):659-662. doi: 10.1053/j.jvca.2022.12.007. Epub 2022 Dec 13. PMID: 36870782.

81 Morine KJ, Kiernan MS, Pham DT, Paruchuri V, Denofrio D, Kapur NK. Pulmonary Artery Pulsatility Index Is Associated With Right Ventricular Failure After Left Ventricular Assist Device Surgery. J Card Fail. 2016 Feb;22(2):110-6. doi: 10.1016/j.cardfail.2015.10.019. Epub 2015 Nov 10. PMID: 26564619.

82 Beshara M, Bagchi A. Dynamic right ventricular outflow tract obstruction: An often-overlooked cause of hemodynamic instability after cardiac surgery. J Cardiothorac Vasc Anesth. 2024;38(9):2035-2040

83. Fortmeier V, Lachmann M, Stolz L, von Stein J, Unterhuber M, Kassar M, Gerçek M, Schöber AR, Stocker TJ, Omran H, Körber MI, Hesse A, Harmsen G, Friedrichs KP, Yuasa S, Rudolph TK, Joner M, Pfister R, Baldus S, Laugwitz KL, Windecker S, Praz F, Lurz P, Hausleiter J, Rudolph V. Artificial intelligence-enabled assessment of right ventricular to pulmonary artery coupling in patients undergoing transcatheter tricuspid valve intervention. Eur Heart J Cardiovasc Imaging. 2024 Mar 27;25(4):558-572. doi: 10.1093/ehjci/jead324. PMID: 37996066.

84. Argiento P, D'Agostino A, Castaldo R, Franzese M, Mazzola M, Grünig E, Saldamarco L, Valente V, Schiavo A, Maffei E, Lepre D, Cittadini A, Bossone E, D'Alto M, Gargani L, Marra AM. A pulmonary hypertension targeted algorithm to improve referral to right heart catheterization: A machine learning approach. Comput Struct Biotechnol J. 2024 Nov 22;24:746-753. doi: 10.1016/j.csbj.2024.11.031. PMID: 39687751; PMCID: PMC11648641.

85. Kadosh BS, Berg DD, Bohula EA, Park JG, Baird-Zars VM, Alviar C, Alzate J, Barnett CF, Barsness GW, Burke J, Chaudhry SP, Daniels LB, DeFilippis A, Delicce A, Fordyce CB, Ghafghazi S, Gidwani U, Goldfarb M, Katz JN, Keeley EC, Kenigsberg B, Kontos MC, Lawler PR, Leibner E, Menon V, Metkus TS, Miller PE, O'Brien CG, Papolos AI, Prasad R, Shah KS, Sinha SS, Snell RJ, So D, Solomon MA, Ternus BW, Teuteberg JJ, Toole J, van Diepen S, Morrow DA, Roswell RO. Pulmonary Artery Catheter Use and Mortality in the Cardiac Intensive Care Unit. JACC Heart Fail. 2023 Aug;11(8 Pt 1):903-914. doi: 10.1016/j.jchf.2023.04.007. Epub 2023 Jun 14. PMID: 37318422; PMCID: PMC10527413.

Echocardiography (Not Right Heart Catheterization or Pulmonary Artery Catheterization) is the Preferred Modality to Assess and Manage Right Heart/Right Ventricular Function

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Over the past 2 decades, a wealth of studies have demonstrated the pivotal role of RV function as a significant predictor of mortality and morbidity across a diverse range of cardiovascular diseases. However, the precise evaluation of RV function continues to present a persistent challenge.[1–3]

The unique anatomy, complex geometry, and unfavorable RV position in the chest cavity make echocardiographic RV assessment a complex task. However, echocardiography's distinct advantages, such as its widespread availability, relative affordability, noninvasiveness, safety, provision of real-time imaging, and rapid results, have solidified its position as the cornerstone of RV assessment in both intra-operative and clinical settings.[4] The unique features of Echocardiography in RV assessment, which set it apart from other methods, can be summarized as follows:

Qualitative Assessment:
Echocardiography provides the advantage of fast and real-time qualitative RV assessment, including the interventricular septum's shape, position, and motion. In overloaded conditions, the normal crescent RV shape would alter by modifying the LV to a "D" shape and flattening the IVS. Visual assessment of IVS flattening timing is a key feature that could help differentiate volume overload (Flattening in diastole) from pressure overload (flattening persists during systole).[5] A visual assessment of RV size can be made in the apical/mid-esophageal four chambers view by comparing it to the LV size and the position of the RV about the apex.

Qualitative evaluation of RV wall motion is also important, as some pathologies present with specific RV motion abnormalities, like the McConnel sign (Hypokinesia of the free wall with maintained apex motion) in Pulmonary embolism[6] and localized RV aneurysm or dyskinesia in arrhythmogenic cardiomyopathy.[7]

Quantitative assessment:
RV size measurements can be obtained from certain views at the end of diastole, with normal values outlined by ASE guidelines. However, avoiding RV foreshortening in the mid-esophageal 4-chamber view is crucial, as this can lead to inaccurate measurements. Adjusting gain settings and compression appropriately can improve image quality and measurement accuracy. Along with RV basal and mid-cavity dimensions, other RV diameters are essential in particular clinical conditions like RVOT for calculating QP/QS for shunts and end-diastole RV thickness at sub-tricuspid regions in RV pressure overload and storage disease.[8]

3D Echocardiography
Despite the RV's intricate anatomy and complex geometry posing a challenge for function assessment with 2D echocardiography, the precision of 3D echocardiography has significantly overcome these limitations, instilling confidence in its accuracy.[4] It allows for the efficient generation of full-volume images with higher temporal and spatial resolution, either through a single beat or stitching multiple volumes in consecutive multi-beat fashion. The 3D dataset can then be analyzed with semi-automatic software providing RV measurements and volumes. In different studies, RV 3D volume data have been proven to be highly reproducible and correlate reasonably well with CMRI and is superior to CT, radionuclide ventriculography, gated single-photon emission CT, radionuclide ventriculography, and invasive cardiac cineventriculography.[9–13]

CON

Despite Shimada et al. 's meta-analysis indicating that 3D echocardiography underestimates the End-systolic volume (ESV), End-diastolic volume (EDV), and Ejection Fraction (EF),[14] it remains a promising modality. Its portability, wide availability, absence of ionizing radiation, and ability to assess patients with defibrillators and pacemakers make it an attractive option, particularly as advancements in technology and methodologies enhance its efficacy.

Conventional RV function assessment parameters

While 3D remained the preferred echocardiographic method of calculating RVEF from RVED and RVES volume, several surrogate echocardiographic indices (TAPSI, RVS', FAC, RV myocardial performance) have also been proposed for clinical use. Often, no single parameter is sufficient, and an integrative approach is required to assess the RV function.[15,16]

RV myocardial deformation imaging

Speckle-tracking echocardiography, an angle-independent technique to evaluate RV longitudinal strain, holds great potential for quantifying myocardial motion in various cardiac conditions, particularly in PAH. Its prognostic and clinical values have been shown in a variety of cardiac pathologies, such as cardiomyopathies, pulmonary hypertension, heart failure, and left ventricle-assisted devices.[17–19]

Global RV longitudinal strain neglects the RVOT and other walls, though, as it averages six segments in the free wall and IVS. Nyberg et al. presented normative data on RVFW longitudinal strain in 1329 healthy individuals from -24 to -16. Even though there is relative agreement on the global strain normal value, the normative data for each segment is still unclear.[20–22]

RV Pressure-Volume loop and End systolic Pressure-Volume relationship analysis

Considering the RV's unique physiology and complex interaction with the pulmonary vascular system, an accurate RV evaluation should include assessing the RV and pulmonary vascular system as a cardiopulmonary unit in a pressure-volume (PV) relationship. RV PV loop analysis involves tracking both pressure and volume over the entire cardiac cycle. While almost all traditional RV echocardiographic assessment parameters are load-dependent, RV PVL analysis addresses the limitations of solely pressure- or volume-based assessments by integrating both factors to create a load-independent measure of RV contractility.[23]

Richter et al. presented a novel non-invasive technique for constructing RV PV loops using echocardiography in patients with PH. This method involves creating an "averaged-out" reference RV pressure curve from pooled pressure wire data and adjusting it according to patient-specific isovolumic and ejection phase durations and echocardiography-derived estimated RV systolic pressure.[24]

Kiarad et al. presented their method of obtaining intraoperative RV pressure-volume (PV) loops in cardiac surgery patients using 3D RV echocardiography and RV pressure data from Transduced RV port of Pulmonary artery catheter. Then, Pressure-time and volume-time curves were synchronized to construct the RV PV loop and extract ESPVR data.[25]

The unique features of echocardiography including ability to assess anatomic features, availability, noninvasiveness, safety, and real-time rapid results, have solidified its position as the cornerstone of RV assessment in clinical setting in contrast to the invasive nature of pulmonary artery catheterization.

1. Amsallem M, Mercier O, Kobayashi Y, Moneghetti K, Haddad F. Forgotten No More. JACC: Heart Failure. 2018 Nov;6(11):891–903.

2. Sanz J, Sánchez-Quintana D, Bossone E, Bogaard HJ, Naeije R. Anatomy, Function, and Dysfunction of the Right Ventricle. Journal of the American College of Cardiology. 2019 Apr;73(12):1463–82.

3. Surkova E, Peluso D, Kasprzak JD, Badano LP. Use of novel echocardiographic techniques to assess right ventricular geometry and function. Kardiol Pol. 2016 Jun 15;74(6):507–22.

4. Valsangiacomo Buechel ER, Mertens LL. Imaging the right heart: the use of integrated multimodality imaging. European Heart Journal. 2012 Apr 2;33(8):949–60.

5. Jurcut R, Giusca S, La Gerche A, Vasile S, Ginghina C, Voigt JU. The echocardiographic assessment of the right ventricle: what to do in 2010? European Journal of Echocardiography. 2010 Mar 1;11(2):81–96.

6. Authors/Task Force Members, Konstantinides SV, Torbicki A, Agnelli G, Danchin N, Fitzmaurice D, et al. 2014 ESC Guidelines on the diagnosis and management of acute pulmonary embolism. European Heart Journal. 2014 Nov 14;35(43):3033–80.

7. Jurcut R, Giusca S, La Gerche A, Vasile S, Ginghina C, Voigt JU. The echocardiographic assessment of the right ventricle: what to do in 2010? European Journal of Echocardiography. 2010 Mar 1;11(2):81–96.

8. Rudski LG, Lai WW, Afilalo J, Hua L, Handschumacher MD, Chandrasekaran K, et al. Guidelines for the Echocardiographic Assessment of the Right Heart in Adults: A Report from the American Society of Echocardiography. Journal of the American Society of Echocardiography. 2010 Jul;23(7):685–713.

9. Lu X, Nadvoretskiy V, Bu L, Stolpen A, Ayres N, Pignatelli RH, et al. Accuracy and Reproducibility of Real-Time Three-Dimensional Echocardiography for Assessment of Right Ventricular Volumes and Ejection Fraction in Children. Journal of the American Society of Echocardiography. 2008 Jan;21(1):84–9.

10. Niemann PS, Pinho L, Balbach T, Galuschky C, Blankenhagen M, Silberbach M, et al. Anatomically oriented right ventricular volume measurements with dynamic three-dimensional echocardiography validated by 3-Tesla magnetic resonance imaging. J Am Coll Cardiol. 2007 Oct 23;50(17):1668–76.

11. Pickett CA, Cheezum MK, Kassop D, Villines TC, Hulten EA. Accuracy of cardiac CT, radionucleotide and invasive ventriculography, two- and three-dimensional echocardiography, and SPECT for left and right ventricular ejection fraction compared with cardiac MRI: a meta-analysis. Eur Heart J Cardiovasc Imaging. 2015 Aug;16(8):848–52.

12. Sugeng L, Mor-Avi V, Weinert L, Niel J, Ebner C, Steringer-Mascherbauer R, et al. Multimodality comparison of quantitative volumetric analysis of the right ventricle. JACC Cardiovasc Imaging. 2010 Jan;3(1):10–8.

13. Zhang QB, Sun JP, Gao RF, Lee APW, Feng YL, Liu XR, et al. Feasibility of single-beat full-volume capture real-time three-dimensional echocardiography for quantification of right ventricular volume: validation by cardiac magnetic resonance imaging. Int J Cardiol. 2013 Oct 9;168(4):3991–5.

14. Shimada YJ, Shiota M, Siegel RJ, Shiota T. Accuracy of right ventricular volumes and function determined by three-dimensional echocardiography in comparison with magnetic resonance imaging: a meta-analysis study. J Am Soc Echocardiogr. 2010 Sep;23(9):943–53.

15. Surkova E, Peluso D, Kasprzak JD, Badano LP. Use of novel echocardiographic techniques to assess right ventricular geometry and function. Kardiol Pol. 2016;74(6):507–22.

16. Surkova E, Cosyns B, Gerber B, Gimelli A, La Gerche A, Ajmone Marsan N. The dysfunctional right ventricle: the importance of multi-modality imaging. Eur Heart J Cardiovasc Imaging. 2022 Jun 21;23(7):885–97.

17. Hardegree EL, Sachdev A, Villarraga HR, Frantz RP, McGoon MD, Kushwaha SS, et al. Role of serial quantitative assessment of right ventricular function by strain in pulmonary arterial hypertension. Am J Cardiol. 2013 Jan 1;111(1):143–8.

18. Hayek S, Sims DB, Markham DW, Butler J, Kalogeropoulos AP. Assessment of right ventricular function in left ventricular assist device candidates. Circ Cardiovasc Imaging. 2014 Mar;7(2):379–89.

19. Lisi M, Cameli M, Righini FM, Malandrino A, Tacchini D, Focardi M, et al. RV Longitudinal Deformation Correlates With Myocardial Fibrosis in Patients With End-Stage Heart Failure. JACC Cardiovasc Imaging. 2015 May;8(5):514–22.

20. Bansal M, Cho GY, Chan J, Leano R, Haluska BA, Marwick TH. Feasibility and accuracy of different techniques of two-dimensional speckle based strain and validation with harmonic phase magnetic resonance imaging. J Am Soc Echocardiogr. 2008 Dec;21(12):1318–25.

21. Fine NM, Chen L, Bastiansen PM, Frantz RP, Pellikka PA, Oh JK, et al. Reference Values for Right Ventricular Strain in Patients without Cardiopulmonary Disease: A Prospective Evaluation and Meta-Analysis. Echocardiography. 2015 May;32(5):787–96.

22. Nyberg J, Jakobsen EO, Østvik A, Holte E, Stølen S, Lovstakken L, et al. Echocardiographic Reference Ranges of Global Longitudinal Strain for All Cardiac Chambers Using Guideline-Directed Dedicated Views. JACC Cardiovasc Imaging. 2023 Dec;16(12):1516–31.

23. Brener MI, Masoumi A, Ng VG, Tello K, Bastos MB, Cornwell WK, et al. Invasive Right Ventricular Pressure-Volume Analysis: Basic Principles, Clinical Applications, and Practical Recommendations. Circ Heart Fail. 2022 Jan;15(1):e009101.

24. Richter MJ, Yogeswaran A, Husain-Syed F, Vadász I, Rako Z, Mohajerani E, et al. A novel non-invasive and echocardiography-derived method for quantification of right ventricular pressure-volume loops. Eur Heart J Cardiovasc Imaging. 2022 Mar 22;23(4):498–507.

25. Kiarad V, Mahmood F, Hedayat M, Yunus R, Nicoara A, Liu D, et al. Intraoperative right ventricular end-systolic pressure–volume loop analysis in patients undergoing cardiac surgery: A proof-of-concept methodology. JTCVS Open. 2024 Dec;22:225–34.