SCA NEWS

SOCIETY OF CARDIOVASCULAR ANESTHESIOLOGISTS
Knowledge • Care • Investigation

Volume 41, Number 24
February 2024

PRESIDENT'S MESSAGE

Kathryn E. Glas MD, MBA, FASE

President, Society of Cardiovascular Anesthesiologists

The Board of Directors and SCA staff completed our winter meeting recently. We spent a weekend discussing our educational offerings and financial planning, including optimizing use of current reserves, and expanding our fund-raising opportunities, as well as support of our research and leadership development missions.

Be on the lookout for Honor a Mentor at the Annual Meeting! Many of us had an attending during residency or fellowship who sparked our interest in this specialty, and then guided us through early career development, and academic or leadership development. For those of you who could not attend last year's gala, this year you have an excellent opportunity to show your mentor how much they mean to you and support SCA development of future leadership, education, and research opportunities through this fund-raising event.

This year's Annual Meeting in Toronto, Canada marks our first ever event in concert with our surgical colleagues from the American Association of Thoracic Surgery (AATS). Drs. Mary Beth Brady, Jonathan Ho and Stephanie Ibekwe have created a first-rate event that includes excellent content and numerous multi-disciplinary sessions with surgery and cardiology colleague engagement. Your registration for SCA grants full access to both the SCA and the AATS programs (excluding extra paid sessions). SCA is honored to have Dr. Jerome Adams, former Surgeon General, as our keynote speaker, and there will be a panel of anesthesiologists, surgeons and cardiologists discussing leadership in the CV care space as well. Note that some hotels are a longer walk from the meeting space than our usual, and we will be providing shuttle services in the morning and afternoon.

Our annual election cycle has begun. All active members are eligible to vote and received an email January 22. Some individuals reported the email went to their SPAM folder, so look there if you have not seen it. Future mailings will change the subject line in hopes of avoiding the SPAM issue. We are fortunate to have numerous excellent candidates for office and encourage each of you to participate in voting for the individuals you feel will best represent your interests and needs on the Board of Directors and CME Committee. The Executive Committee and Board of Directors welcomes your feedback on how we can better represent your needs as a CVT Anesthesiologist.

Thanks to Dr. Anne Cherry from the Research Committee diligently collating information on the success of our research grant. Since 2008, our Society has funded early career, mid-career and MiCOR grant winners with almost 3 million dollars. These funds have led to 81 publications documenting new knowledge in cardiovascular disease and improved patient care. As important is the impact of these grants on their ability to generate federal funding to further knowledge generation. Almost 17 million in federal funding has been received by researchers using society funding mechanisms. Each of us benefits from our support of these projects, and ultimately our patients do as well.

Best Regards,

Kathryn E. Glas MD

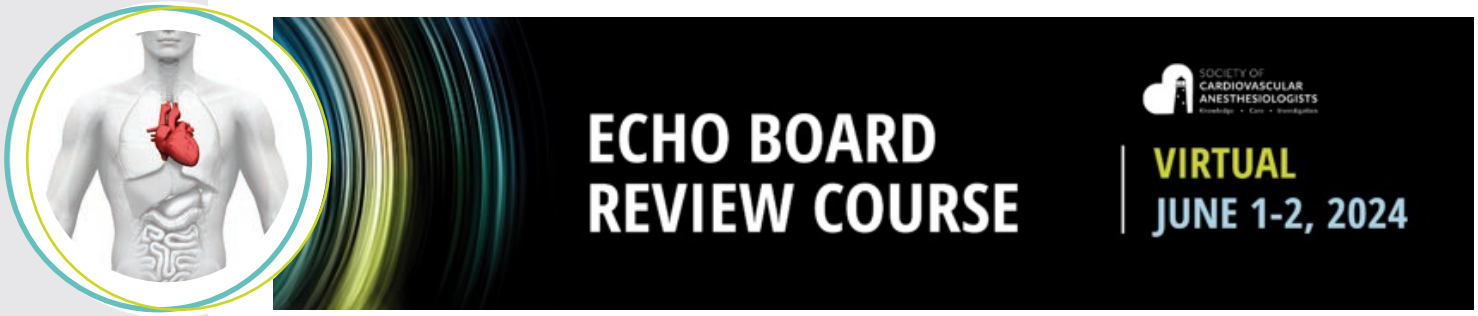
Missed 2024 Echo Week?

Even if you were unable attend to the 2024 Annual Echo Week, that does not mean you have to miss out on valuable content!

Echo Week will be recorded and provide the opportunity to deepen your understanding of ultrasound and perioperative transesophageal echocardiology with access to nationally recognized experts and content that will enhance your practice. You can access the product anytime, anywhere—all while earning CME credits!

Whether you were unable to attend in-person or virtually or want to revisit sessions you missed at the meeting, Echo Week recorded is just what you need. **Details are forthcoming on how to obtain ECHO WEEK 2024.**

Join Us to Prepare for the Echo Board Exam Review Course



A panel of experts will lead sessions designed to help prepare Echo Board candidates for the exam. The Echo Board Exam Review Course is designed for Fellows who will be sitting for the exam for the first time and for those who will be taking the exam to recertify their credentials.

AGENDA COMING SOON

REGISTRATION OPENS, MONDAY, FEBRUARY 26, 2024

Register for COR-PM Today!

April 27-29, 2024 — Toronto, Canada

The Scientific Program Committee is thrilled to announce the third Cardiovascular Outcomes Research in Perioperative Medicine (COR-PM) conference will be held in person April 27-29, 2024, in conjunction with the SCA 46th Annual Meeting and Workshops in Toronto, Canada.

REGISTER NOW VIEW AGENDA

Introducing Our Keynote Speaker

COR-PM 2024

Beverley Anne Orser

MD, PhD, FRSC, FCAHS, FRCPC

Preserving Brain Function After Surgery

Dr. Orser is the Professor and Chair of the Department of Anesthesiology & Pain Medicine, Temerty Faculty of Medicine, University of Toronto, and the Chair of the Board of the International Anesthesia Research Society. She is also a practicing anesthesiologist at Sunnybrook HealthSciences Centre in Toronto, Canada. Her clinical studies and advocacy work focus on patient safety and the anesthesia workforce in Canada. She has co-founded several patient safety organizations, including ISMP Canada, the Canadian Anesthesiologists' Society Quality & Patient Safety Committee, and the Perioperative Brain Health Centre. Her preclinical research has offered fundamental insights into the molecular basis of anesthesia, including the longer-term effects of exposure to anesthetic drugs. Her laboratory first demonstrated the unique pharmacological properties of extra synaptic GABAA receptors and identified novel roles for these receptors in health and disease. Her advocacy work has improved patient care and her discoveries have led to patents and potential new treatments.

Join Us at TAS in April!

Register NOW!

The Thoracic Anesthesia Symposium (TAS) Planning Committee invites you to join the world of non-cardiac anesthesiologists from around the world for the 2024 TAS meeting on April 26, 2024, in Toronto, Canada.

Look forward to:

- A focus on dramas, traumas, along with everyday challenges in thoracic anesthesiology.
- Exploration of the latest literature and current controversies by international experts in the field.
- Hands-on workshop featuring new and updated workshop stations with live models, custom high-fidelity 3D phantom models, and 3D anatomic visualization!

At the SCA Thoracic Anesthesia Symposium you can:

- Choose 3 in-person workshops and register for an optional live PBLD for a conference experience tailored to YOUR educational needs.
- Network with 200 other professionals in anesthesiology to help you gain insight into your practice and career.
- Connect with our exhibitors to learn about new products and programs.

REGISTER NOW VIEW AGENDA

TAS Problem Based Learning Discussions (PBLDs)

Saturday, April 27, 2024
7:00 AM – 8:00 AM

PBLD #1 -	Lung Transplantation in Kartagener's Syndrome - Mirror, Mirror, on the Wall
PBLD #2 -	Setting up a Regional Anesthetic Service for Thoracic Surgery
PBLD #3 -	Massive Hemoptysis in Interventional Pulmonology Suite
PBLD #4 -	It's Only a Simple Procedure – Segmentectomy in a Patient with Severe Pulmonary Hypertension and Right Ventricular Dysfunction
PBLD #5 -	Anesthetic Management for a Patient with an Anterior Mediastinal Mass – When DoYou Need Backup?
PBLD #6 -	Challenges in Acute Pain Management: The Patient with Cancer Undergoing Minimally Invasive Esophageal Surgery

Don't Delay! Register Today!

Register NOW for the 2024 Annual Meeting & Workshops!

SCA 2024 Annual Meeting and Workshops in collaboration with AATS

Please join us at the Society of Cardiovascular Anesthesiologists 46th Annual Meeting and Workshops in beautiful Toronto, Canada, April 27-30, 2024.

For the first time ever, the SCA Annual Meeting will be presented in collaboration with the American Association for Thoracic Surgeons (AATS). We are pleased to announce that registration to the SCA Annual Meeting grants full access to the entire AATS program (except for paid sessions). You will not want to miss this exciting educational opportunity!

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AATS

Registration to the SCA Annual Meeting grants full access to the entire AATS program except for paid sessions such as workshops! View the AATS program!

PROFESSIONAL DEVELOPMENT WORKSHOP

Sunday, April 28, 2024 | 9:00 AM – 12:00 PM

Achieving Success in Cardiac Anesthesia: Skill-building Workshop for Professional Development and Career Advancement, and Leadership

Description: This unique, interactive workshop will integrate expertise from both the academic and business worlds to help SCA members navigate and succeed in both the academic and private practice landscape, with the goal of fostering future leaders. Specifically, attendees will work on skill development in networking, mentorship, negotiation, and presentation. Take-home lessons include how to perfect the "elevator pitch," cultivate healthy mentor and sponsor relationships, negotiate for time and compensation, and create and deliver an effective presentation.

Moderators: Dalia Banks, MD, University of California, San Diego; Emily Methangkool, MD, University of California, Los Angeles; and Sarah Smith, MD, MS, Westchester Medical Center

What I Look for in Leaders: How to Choose the Right Person for the Right Task
Peter Panzica, MD, Westchester Medical Center

How to Deliver a Mind-blowing Presentation:
Emily Methangkool, MD, MPH, University of California, Los Angeles

Conflict Management: How to Ease Egos and Tame Tempers in the Operating Room
Sasha Shillcutt, MD, University of Nebraska Medical Center

When to Say Yes: Optimizing Opportunities for Career Development
Timothy Maus, MD, FASE, University of California San Diego

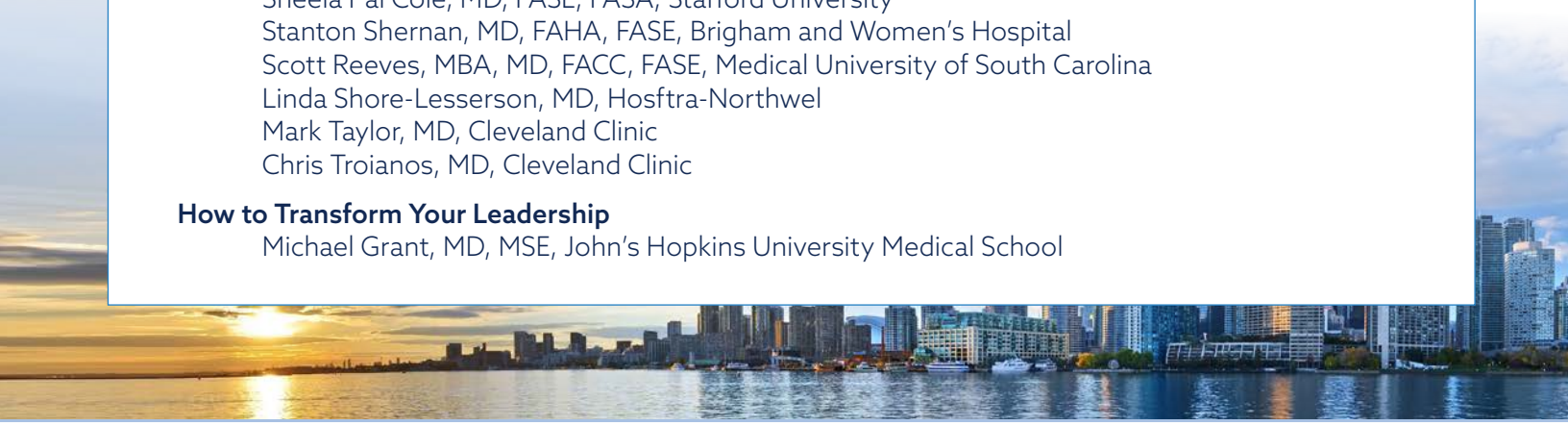
Small Group Moderators:
Negotiating Tips
Thomas McLoughlin, MD, Jr., FASA, Lehigh Valley Health Network
Douglas Shook, MD, FASE, Brigham and Women's Hospital

Networking 101 - How to Perfect Your "Elevator Pitch"
Jonathan Leff, MD, Montefiore Medical Center

1:1 Mentoring for Career Development with SCA Leaders
Mentors:
Natalia Ivascu Girardi, MD, Weill Cornell Medicine
Kathryn Glas, MD, MBA, University of Arizona College of Medicine, Tucson
Jacques Prince Neelankavil, MD, University of California, Los Angeles
Sheela Pai Cole, MD, FASE, FASA, Stafford University
Stanton Shernan, MD, FAHA, FASE, Brigham and Women's Hospital
Scott Reeves, MBA, MD, FACC, FASE, Medical University of South Carolina
Linda Shore-Lesserson, MD, Hosftra-Northwel
Mark Taylor, MD, Cleveland Clinic
Chris Troianos, MD, Cleveland Clinic

How to Transform Your Leadership
Michael Grant, MD, MSE, John's Hopkins University Medical School

Space is Filling Up Fast for This Session!



Register NOW

Problem-Based Learning Discussions (PBLD)

Still Time to Register Before Space Fills Up!

Space Still Available for PBLD #1 through PBLD #6 — Saturday, April 27, 2024 | 7:00 AM – 8:00 AM

PBLD 9

Mitral Regurgitation: A Team-Based Approach to Decision-Making

Regina Linganna, MD
University of Pennsylvania Health System

Aidan Sharkey, MD
Beth Israel Deaconess Medical Center

PBLD 10

VAD Emergencies

Christina Jelly, MD
Vanderbilt University Medical Center

Jenny Kwak, MD, FASE
Loyola University Medical Center

FELLOW AND RESIDENT PROGRAMS

Workshops enables Fellows and Residents to attend incredible educational sessions specifically designed for the trainee.

POSTER SESSIONS:

Fellow and Resident Posters: Session 1
Saturday, April 27 | 11:00 AM - 12:00 PM

Fellow and Resident Posters: Session 2
Saturday, April 27 | 4:00 PM - 5:00 PM

Fellow and Resident Posters: Session 3
Sunday, April 28 | 8:00 AM - 9:00 AM

Fellow and Resident Program: Mission Possible
Saturday, April 27 | 1:00 PM - 2:00 PM

Fellow and Resident Program: Echo Essentials for the Emerging Professional
Saturday, April 27 | 2:00 PM - 3:30 PM
Moderators:
Tara Brakke, MD, University of Nebraska Medical Center
Jennifer Hargrave, DO, Cleveland Clinic

Understanding of Intra Atrial Shunts
Kiran Belani, MD, FASE, FACC, Northwestern University

Hypertrophic Cardiomyopathy for the Masses
Candice Morrissey, MD, MSPH, FASE, University of Utah

Predicting SAM Post Mitral Valve Repair
Stanton Shernan, MD, FAHA, FASE, Brigham and Women's Hospital

REGISTER NOW

AS A MENTEE

Saturday, April 27 | 5:00 PM - 6:00 PM

Fellow and Resident Mentor/Mentee Session

SCA2024 ANNUAL MEETING & WORKSHOPS

Call for Mentees is Now Open!

As a mentee, you have chosen to take an important step in actively shaping and planning a career in cardiac anesthesiology, which is critical for the success and development as a faculty member. This program consists of the following:

- Getting to know your mentor, building a relationship, and establishing needs and expectations.
- Completing a personal faculty assessment (PFA), which can provide a very useful link between your starting point and eventual goals (assessment will be sent later).
- Discussing your personal and professional goals with your mentor, who will help you to identify one or two achievable goals and develop a plan.
- Important topics for future communication might include new projects, career development, upcoming deadlines, and personal/professional updates.
- Mentors and mentees will be paired in late March. We will be back in touch in the spring with your match.

NOTE: This program will be held during the 2024 Annual Meeting in Toronto, Canada and requires your in-person attendance at the Mentor/Mentee Reception.

REGISTER NOW

VIEW PROGRAM

Sunday, April 28 | 10:45 AM - 1:45 PM

Fellow and Resident Review Course: Keys to Success: High Yield Topics for the Adult Cardiac Anesthesiology Exam

Moderators: Tara Brakke, MD, University of Nebraska Medical Center and
Jennifer Hargrave, DO, Cleveland Clinic

Mitral Stenosis/Insufficiency
Abimbola O. Faloye, MD, FASE, FASA
Emory University

Electrophysiologic Disturbances
Reed Harvey, MD
University of California, Los Angeles

Circulatory Assist Devices
Jeffrey Songster, MD
University of Nebraska Medical Center

Congenital Heart Disease
Nelson Burbano-Vera, MD
Cleveland Clinic

Aortic Stenosis/Insufficiency
Brandi Bottiger, MD
Duke University School of Medicine

Rare Cardiac Diseases
Megan Kostibas, MD
The Johns Hopkins School of Medicine

Sunday, April 28 | 3:00 PM - 5:30 PM

Early Career Investigator Awards

Moderators: Anne Cherry, MD, FASE, Duke University
Jonathan Ho, MD, University of California, Los Angele
Lisa Rong, MD, MSCE, FASE, FACC, New York-Presbyterian Weill Cornell Medicine

Introducing Our Earl Wynands Lecturer

Louise Sun, MD, SM, FRCPC, FAHA
Stanford University

Through the Center of Cardiovascular Research: My Journey with Big Data and Bioengineering

Dr. Sun is Professor and Chief of Stanford Cardiothoracic Anesthesiology. Her patient-centered research program leverages big data to bridge key gaps in healthcare delivery and outcomes for patients with heart failure and those undergoing cardiac procedures. She specializes in rapid deployment of data-driven solutions to enhance operational efficiency and patient care. Dr. Sun sits on several editorial boards and scientific review committees, and has authored over 100 peer-reviewed publications, many in leading journals including JAMA, Circulation, JACC, and Diabetes Care.

Introducing Our Keynote Speaker

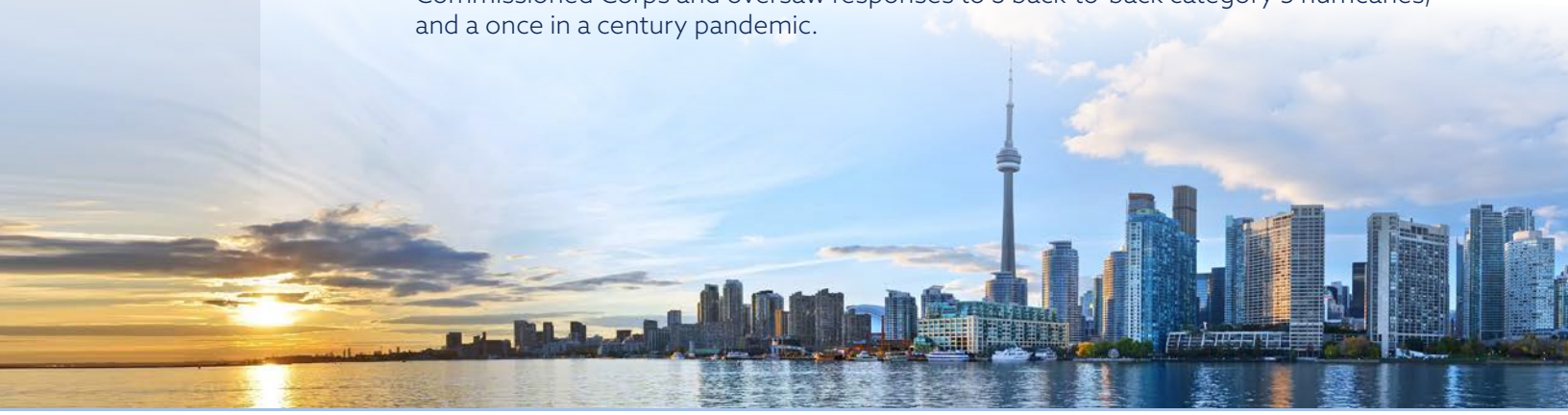
Jerome Adams, MD, MPH, FASA

Health Equity: What Is It? Why Does It Matter?

Dr. Jerome Adams was appointed as a Presidential Fellow and the Executive Director of Purdue's Health Equity Initiatives on October 1, 2021. He is also a Distinguished Professor of Practice in the departments of Pharmacy Practice and Public Health.

As the 20th U.S. Surgeon General and a prior member of the President's Coronavirus task force, Dr. Adams has been at the forefront of America's most pressing health challenges. A regular communicator via TV, radio, and in print, Dr. Adams is an expert not just in the science, but also in communicating the science to the lay public and making it relevant to various audiences.

Dr. Adams is a licensed anesthesiologist with a master's degree in public health and ran the Indiana State Department of Health prior to becoming Surgeon General. In the State Health Commissioner role, he managed a $350 million dollar budget and over 1,000 employees, and led Indiana's response to Ebola, Zika, and HIV crises. Notably, Dr. Adams helped convince the Governor and State Legislature to legalize syringe service programs in the state, and to prioritize$13 million in funding to combat infant mortality. As Surgeon General, Dr. Adams was the operational head of the 6,000-person Public Health Service Commissioned Corps and oversaw responses to 3 back-to-back category 5 hurricanes, and a once in a century pandemic.



ASA Presidential Update

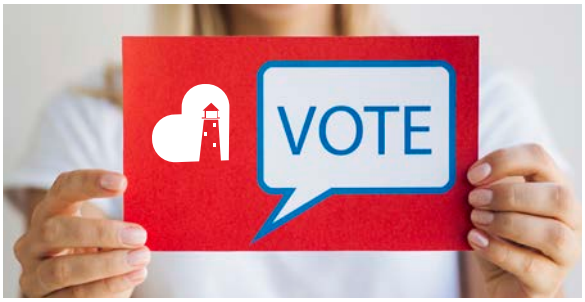
Ronald Harter, MD, FASA
The Ohio State University
Wexner Medical Center

Ronald L. Harter, M.D., FASA, professor of anesthesiology in the Department of Anesthesiology at The Ohio State University Wexner Medical Center in Columbus, was today named president of the American Society of Anesthesiologists (ASA), the nation's largest organization of physician anesthesiologists. Dr. Harter assumed office at the ANESTHESIOLOGY® 2023 annual meeting and will serve for one year.

Dr. Harter is a member of ASA's Executive Committee and Administrative Council. Over the past three decades, he has served ASA in numerous roles, most recently as ASA president-elect. He served as speaker of ASA's House of Delegates and ASA's representative to the National Academy of Medicine's Action Collaborative on Clinician Well-Being and Resilience. He has chaired ASA's Ad Hoc Committee on Physician Well-Being, Committee on Young Physicians, Committee on Medical Students and Residents, and Committee on Bylaws.

Additionally, Dr. Harter has been an active member of the Ohio Society of Anesthesiologists since 1994 and served as its president in 2003.

Dr. Harter received a Bachelor of Arts in chemistry from Capital University in Bexley, Ohio, and completed his Doctor of Medicine at The Ohio State University in Columbus. He completed his residency in anesthesiology at Georgetown University Medical Center in Washington, D.C., and is board-certified by the American Board of Anesthesiology.



SCA 2024 Elections

VOTING IS NOW OPEN!

The 2024 online elections for SCA leadership positions are open through March 11. The candidates are running for the following positions:

Director-at-Large

Early Career Director

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 2024 election cycle:

Director-at-Large Candidates

Tara Brakke, MD, FASE

University of Nebraska Medical Center

I have been on faculty at the University of Nebraska Medical Center in Omaha, Nebraska since 2004. I have enjoyed serving as the Chief of Cardiovascular Anesthesiology since 2007 and am particularly proud of the growth of our team and the care we provide the region. As the inaugural program director for the Adult Cardiothoracic Anesthesiology fellowship from 2012 to 2020, I appreciated all the educational opportunities the SCA provides its' members. Within the SCA, I have been honored to participate on the Continuing Medical Education Committee, the Scientific Program Planning Committee, Co-Direct the fellow and resident program, and the Endowment Council. I have also thoroughly enjoyed serving on the Board of Directors the past three years and would love the opportunity to continue to represent the membership and the future of our society in this role.

Abimbola (Bola) Faloye, MD, FASA, FASE

Emory University School of Medicine

Dr. Bola Faloye is an Associate Professor in the division of cardiothoracic anesthesiology, Emory University School of Medicine. She currently serves as the Director of Mentors in Medical Education Program for the Department of Anesthesiology, and previously served as the Division Chief of Adult Cardiothoracic Anesthesiology at Grady Memorial Hospital. Dr. Faloye is active within several national organizations including serving as the Chair of SCA-WICTA (Women in Cardiothoracic Anesthesia) Special Interest Group, a member of the SCA Scientific Program Planning Committee. She also serves on the ASA Educational Track Subcommittee on Cardiac Anesthesia, and the ASE Council on Perioperative Echo. Her area of research focuses on mechanical circulatory support in adults with heart failure. She is an expert in perioperative echocardiography and routinely lectures in this area nationally and internationally. She has authored numerous manuscripts and textbook chapters on cardiac anesthesiology and perioperative echocardiography.

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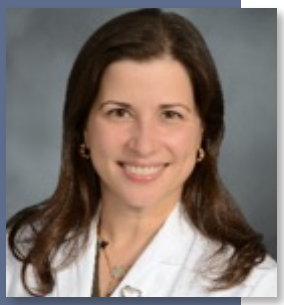
Director-at-Large Candidates



Natalia Ivascu Girardi, MD

Weill Cornell Medicine

Natalia Ivascu Girardi, MD is Professor of Clinical Anesthesiology and Professor of Medical Ethics in Clinical Medicine, at Weill Cornell Medicine in New York City. She is Executive Vice Chair for Clinical Affairs in the Department of Anesthesiology, and Chief of Critical Care Anesthesiology. Dr. Girardi graduated from University of Michigan and Wayne State University School of Medicine. She completed residency and fellowship training at New York-Presbyterian Hospital/Weill Cornell Medicine and Columbia. She is board certified in Anesthesiology and Critical Care Medicine. She is a Diplomate of the American Board of Echocardiography, with certification in Advanced Perioperative Transesophageal Echocardiography and Special Competence in Critical Care Echocardiography. Dr. Girardi's research interests include perioperative bleeding, patient blood management and medical ethics. She previously served two terms on the SCA Ethics Committee. She is currently a member of the SCA Scientific Program Planning Committee (SOCCA Liaison) and Patient Blood Management Committees.

Alina Nicoara, MD

Duke University

Having been a member of the SCA since 2006, I have had the privilege to contribute to its growth through various committees and task forces, including the SCA Scientific Program Planning Committee, Nominating Committee, STS/SCA Database Sub-Committee, and the Cardiac Anesthesia Board Certification Task Force, among others. My roles within the SCA, complemented by responsibilities as a speaker, panelist, and moderator, have allowed me to impart and absorb knowledge, making every interaction a source of inspiration. I am firmly rooted in the philosophy that excellent results, whether in clinical care or academic pursuits, arise from collaborative teams that traverse specialties and academic thresholds. My diverse engagements spanning clinical practice, research, education, administration, and service, resonate with this philosophy. As I seek the position of Director-at-Large, I am eager to spearhead initiatives, fostering growth for our society and specialty at large.

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.

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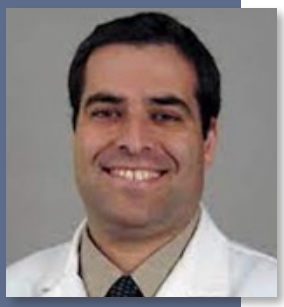
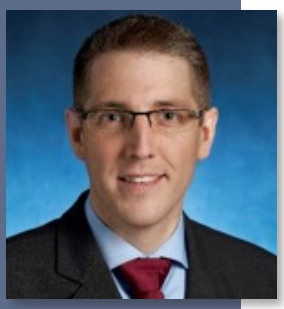
Director-at-Large Candidates



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.

Early Career Director-at-Large

Farzad Ebrahimi, MD

Advocate Illinois Masonic Medical Center

I am Farzad Ebrahimi and I embarked on my medical education at Tehran University of Medical Sciences. In 2008, I joined Harvard Medical School, dedicating two years to medical research. My anesthesiology journey began with a clinical internship at Massachusetts General Hospital, followed by an anesthesiology residency at Advocate Illinois Masonic Medical Center, which is affiliated with University of Illinois at Chicago, where I graduated with distinction in 2014. I specialized in cardiothoracic anesthesiology at Yale University. At Advocate Aurora Health Care I earned the title of Assistant Professor of Anesthesiology. I have mentored residents. As Director of the Simulation Lab, I earned awards and collaborated with SCA's Simulation SIG. I played a role in launching TAVR and mitral clip programs. My research in speckle tracking echocardiography resulted in publications in peer-reviewed journals. I have presented at the SCA and recently for a grand round lecture at Cook County Hospital.

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Early Career Director-at-Large

Regina (Gina) Linganna, MD

University of Pennsylvania

Gina Linganna is an assistant professor at the Hospital of the University of Pennsylvania. She has been an active SCA member since 2016. Her most recent contribution to the SCA is as the creator of ARC: A Review Course – the SCA's board review course for the upcoming cardiac anesthesia board exam. Additionally, she currently chairs the Member Engagement Committee. As chair she has worked with the Women in Cardiothoracic Anesthesiology Special Interest Group (WICTA) to produce several webinars directed at fellows and junior faculty focused on professional development. She is also a member of the Online Education Sub-Committee and WICTA. She was the previous inaugural chair of the SCA Mobile App Sub-Committee and creator of the SCA's mobile application. Gina hopes to be elected to the Board of Directros as an early career member to continue her SCA contributions on a larger scale.

Jessica Spence, MD, PhD, FRCPC

McMaster University

Jessica Spence is a cardiac anesthesiologist and intensivist at McMaster University who completed fellowship training at the University of Toronto in January/2021. Since 2020 she has been actively involved in the SCA as a member of the Research and STS Database Sub-Committees. Despite her early career stage, Dr. Spence is an accomplished researcher whose research program is dedicated to advancing the profession by providing cardiovascular anesthesiologists with the practical clinical practice information they need to guide their daily work, which is in direct alignment with the mission of the SCA. She is the Principal Investigator of the recently completed 20,000 patient B-Free trial, has published more than 70 manuscripts, and holds more than $5 million in funding as Principal Investigator. If elected, she will work tirelessly to ensure that the educational activities of the SCA address both nuanced and emerging areas of practice, but also provide evidence-based guidance to our everyday work.

Agnieszka Trzcinka, MD, FASE

Tufts Medical Center

Dr. Agnieszka Trzcinka completed her anesthesiology residency and cardiac anesthesiology fellowship at the Brigham & Women's Hospital (BWH). She worked as a cardiac anesthesiologist for 3 years at BWH and was recruited to Tufts Medical Center in 2018. She has served in several leadership capacities in the Department of Anesthesiology, the Tufts Physicians Organization, and the Tufts University School of Medicine. Dr. Trzcinka has launched several multidisciplinary projects as Director of Cardiovascular Center Procedural Anesthesia. She served as Chair of the Clinical Practice Committee and Chair of the Salary and Benefits Subcommittee within the Tufts PO Women in Medicine and Science Committee to ensure equality and support for women in the workplace. Dr. Trzcinka serves on the Tufts University Faculty Senate and on the Executive Board of WICTA (Women in Cardiothoracic Anesthesia) Special Interest Group within the SCA. She is a member of the SCA Ethics and Scientific Program Planning Committees.

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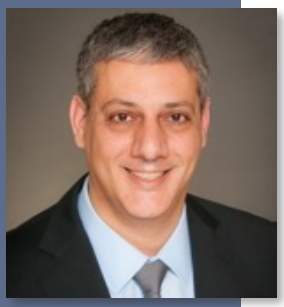
Continuing Medical Education (CME) Committee Member Candidates



Christos Koutentis, MB, ChB, MS

SUNY Downstate Medical Center

Currently, I'm an Assistant Professor in Anesthesiology at SUNY Downstate, and work at an Academic Level 1 trauma center in Brooklyn. Over the last 22 years, I have worked across a variety of settings within the VA system, Private Practice and Academia. My training includes an MS in Epidemiology at Mailman School of Public Health, Anesthesia Residency at Cornell, Cardiac Anesthesia Fellowship at Johns Hopkins, and Medical School at Aberdeen University in Scotland. My Research interests include AKI, Sepsis, Vasoplegia, Transfusion, Neurocognitive Outcomes, and evaluating monitors. Over the last 2 years I have served on the SCA AKI Sub-Committee and am particularly motivated in putting together projects involving MPOG and STS datasets. I was a founding member of the Trauma Anesthesia Society, and serve on committees for the IARS, Society for Technology in Anesthesia, and Society for the Advancement of Transplant Anesthesia. Outside interests include cave diving and wreck exploration.

Eligible voting members received a personalized link for the online election system via email. If you did not receive this email and you believe this to be an error, please contact Denise Herdrich at dherdrich@veritasamc.org.

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2025 SF Match Fellowship Match Program

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. The schedule for the 2025 training year is as follows:

November 6, 2023	Applicant Registration Began
March 6, 2024	Central Application Service Target / Deadline Date
June 5, 2024	Program Rank List Submission Deadline (12 PM PT)
June 5, 2024	Application Rank List Submission Deadline (12 PM PT)
June 19, 2024	Match Results
June 20, 2024	**Post**-Match Vacancies Posted
July 2025	Training Position Starts

Applicants and programs participate by registering with SF Match and applicants applying to the programs of their choice. Both programs and applicants submit a rank list based on theirpreferences. Notably, only programs where an applicant has interviewed can be ranked in the match.

Critical to the match process, programs and applicants can make an Exception Agreement prior to submitting their rank list to SF Match. Exception Agreements allow an applicant and program to agree to match each other prior to submitting their respective rank lists. Importantly, all ACTA positions must be included in the match, including all Exception Agreement positions.

Exceptions to the standard match process have been agreed upon by the ACTA Fellowship Program Directors Council in the following situations:

1. Applicants who are in active military service at the time of application.
2. Internal candidates, i.e., applicants who are currently in the anesthesiology residency program at the same institution as the ACTA fellowship.
3. Applicants who are making a commitment to come to the institution of the ACTA fellowship for more than one year.
4. Applicants who are enrolled in an anesthesiology residency outside of the USA at the time of application.
5. Applicants who reside outside the USA at the time of application or who are not eligible for ABA certification due to non-US training.
6. Applicants whose spouse or partner is applying for a GME-approved post graduate training program in a medical specialty in the same region as the ACTA fellowship.

Please Note: Eligible applicants and programs who wish to take advantage of an exception rule are still required to participate in the match ranking process and must complete an exception agreement found on the SCA website via the link below. This year exception agreements will be posted on the SCA website for transparency to programs and applicants. Any match irregularities will be referred to the ACTA Fellowship Program Directors Council of the SCA.

Program directors complete the first part of the match exception process. **To begin, CLICK HERE.** Please note, you will need to log in with your SCA username and password. Once the program director completes this portion of the process, the applicant will receive an email with a link to the form they must complete. Any match irregularities will be referred to the ACTA Fellowship Program Directors Council of SCA.

For questions or assistance, please contact Mary Lunn at **mary@veritasamc.com.**

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MEMBER CORNER

AWEsome Woman Interview

Benu Makkad, MD

Associate Professor
University of Cincinnati College of Medicine

I am an Associate Professor in the Department of Anesthesiology at the University of Cincinnati College of Medicine, where I have practiced for the past several years. I have truly enjoyed providing care to complex patients undergoing various cardiac and non-cardiac surgical procedures. I also have the privilege of working with medical students, residents, and fellows and mentoring them in their career pursuits. I have had the opportunity to serve as an adult ACTA program director and Director of Cardiac Anesthesia at my institution. I am currently Secretary-Treasurer of the Ohio Society of Anesthesiologists (OSA) and serve on its board.

1. What led you to become a Cardiovascular/Thoracic Anesthesiologist?
During my residency training, I really enjoyed providing care for critically ill surgical patients undergoing cardiac and non-cardiac surgery. During my cardiac anesthesia rotation, I loved cardiopulmonary physiology and was fascinated by the invasive hemodynamic monitoring and perioperative echocardiography used to guide surgical decisions. Every case was different and required quick decision-making and problem-solving skills as well as the teamwork between surgeons, anesthesiologists, perfusionists, and nurses.

2. How did you hear about the SCA?
I learned about the SCA during my fellowship at the Cleveland Clinic, and I have been actively involved since then.

3. What roles have you held for the society?
I have served on the ACTA Fellowship Program Director's Council, where I had the opportunity to network with other program directors and learn from them.

Recently, I had the opportunity to serve on the Presidential Task Force for the Development and Publication of Clinical Practice Parameters, Reviews, and Meta-analyses. I have served on the SCA Quality Safety and Leadership Committee, and I had the opportunity to chair the Opioid Working Group/Task Force. I have moderated many residents' and fellows' abstract poster sessions and participated in regional workshops at the annual SCA meetings. I look forward to contributing to any new initiatives that the SCA may undertake in the future.

4. What is one of your greatest achievements as a Cardiovascular/Thoracic Anesthesiologist?
Getting the Adult ACTA fellowship established and accredited as well as keeping it afloat with the cardiac surgical turnover at our hospital was one of my greatest successes as a cardiothoracic anesthesiologist. Working with a multidisciplinary team to set up the structural heart disease program at our institution is another accomplishment I am really proud of.

I also feel privileged to have led the Opioid Task Force in developing practice advisories to improve the perioperative pain management of cardiac and thoracic surgical patients.

5. Do you have any advice for fellows and residents?
Work hard and learn as much as you can from the staff and other resources available to you at your institution. Set goals to achieve every day with each case you do and each rotation you are assigned to. Actively seek feedback as it provides an opportunity to improve yourself. Build friendships and lend a helping hand to your colleagues and coworkers. Being open, humble, and respectful may open doors for you in the future.

6. Have you experienced any difficulties as a woman in the field?
Yes, I have experienced many challenges and obstacles as a female leader in anesthesiology, and I still do. Unlike male counterparts, women are judged more harshly and are often overlooked for opportunities for their academic growth. But these obstacles have made my

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determination to reach my goals even stronger. When one door was closed, I already had a window of opportunity open and worked twice as hard to achieve my goals. I have been fortunate to have people who have supported my work and helped me reach the finish line.

7. Do you have any advice for other women in the field?
Find your passion early and set a 1–5-year plan to achieve your goal. Seek out mentors and sponsors within your institution, your professional society, or your network of peers and colleagues who will help you navigate these challenges and advocate for your advancement.

Be supportive of other female colleagues by being a mentor or sponsor and helping them achieve their career aspirations. Explore opportunities outside your department at an institutional level and work with local and national societies for the advancement of your field.

Be proud of your accomplishments and share them with others. Recognize and appreciate the achievements and contributions of other women in the field.

8. How do you balance work and personal life?
Work-life balance is an important aspect of any career, especially when you work in a demanding and stressful environment. I prioritize tasks based on their urgency and importance and try to establish clear boundaries between my work and personal life, but it has always been a work in progress.

I am fortunate to have an understanding family and friends who support me in my endeavors. Over time, I have learned to leave the guilt and regrets behind and simply do the best I can.

9. What is something you enjoy doing outside of work?
Outside of work, I enjoy spending time with my family. I also like to take walks, listen to music, and cook.

10. Would you change anything about the path you took to get to where you are now?
I wished I had searched for a mentor or sponsor earlier in my career who could have helped me focus on my goals and career aspirations. But it is never too late to find one.

11. What was the best piece of advice you received?
Be yourself and do not compromise your integrity. Do not let closed-minded people define your path; make your own. Work hard and try your very best. If you fail, get up and try again.

Build friendships and make allies wherever you go.

Echocardiographic Versus Invasive Aortic Valve Gradients in Different Clinical Scenarios

Amr E. Abbas, MD, Houman Khalili, MD, Luai Madanat, MD, Sammy Elmariah, MD, Francis Shannon, MD, Karim Al-Azizi, MD, Thomas Waggoner, MD, Thomas Pilgrim, MD, Taishi Okuno, MD, Anthony Bavry, MD, Julien Ternacle, MD, Jared Christensen, MD, Josep R. Cabau, MD, Michael Mack, MD, and Philippe Pibarot, PhD, DVM

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In a multicenter study, the authors compared mean transaortic valve gradients before and after transcatheter aortic valve replacement (TAVR) obtained with Doppler echocardiography (non-invasive) using the simplified Bernoulli equation, and invasively using intracardiac catheter placement and withdrawal.[1] Absolute discordance and percentage of discordances > 10 and 20 mmHg were recorded. Additional analysis sought to determine variables associated with discordance.

Absolute discordance was calculated as echocardiographic mean gradient minus invasive mean gradient. Percent discordance was calculated as:

$$\frac{\text{(Echo mean gradient – Invasive mean gradient) x 100}}{\text{Echo means gradient}}$$

Aortic valve area (AVA) was calculated using the continuity equation combining Doppler echocardiography and two-dimensional determination of the subvalvular area. AVA was indexed per the patient's body surface area.

Indexed aortic valve area (iAVA) was recorded in 3526 patients and severe patient prosthesis mismatch was reported in 650 patients. Flow states (indexed stroke volume; SVI) was recorded in 1889 and low flow (< 35 ml/min/m2) was reported in 864 patients.[1]

Of 2547 patients with a recorded ejection fraction, 496 had EF < 50%. Aortic valve insufficiency was reported in 4703 patients.[1]

5,027 patients were included of which 4725 had TAVR of native valve (N-TAVR) and 302 had a valve in valve TAVR (ViV-TAVR). Post procedure mean gradients were obtained invasively in 2545 and echocardiographically in 4711.[1] Concomitant invasive and echocardiographic mean gradients were obtained pre-procedure in N-TAVR (n=2418), ViV-TAVR cases (n=101), and post procedure in N-TAVR (n=823) and ViV-TAVR (n=77) valves. Data were collected within 5-10 minutes of each other.[1]

For the 5027 TAVR cases the mean post TAVR invasive mean gradient in 2545 patients was 6mmHg (+ 5) with 35 (1%) having a mean gradient > 20mmHg.[1]

Prior to TAVR there was no significant difference and a good correlation ($r=0.69$; $p < 0.001$) between invasive and non-invasive mean transvalvular gradients.[1] After TAVR there were significant differences between mean invasive and echocardiography gradients for both N-TAVR (0 vs 4 mmHg resp.) and ViV-TAVR (4 v 10mmHg resp.), the former with poor correlation compared to the latter ($r = 0.18$ and $r = 0.61$ respectively).[1] Mean post TAVR bias between invasive and echocardiography was 3.41 (-3.62 to 10.53) mmHg for N-TAVR cases, and 6.47 (-5.51 to 18.44) mmHg for ViV-TAVR. Post procedure discordance > 10 mmHg was present in 39 N-TAVR (5%), and 60 ViV-TAVR patients (27%).[1] Independent predictors of discordance included ViV-TAVR, younger age, and smaller valve size. Absolute discordance increased with increasing echocardiographic mean gradient e.g. > 20 mmHg.[1]

At discharge, the mean echocardiographic gradient (n= 4711) was 10mmHg (+ 5) with 279 (6%) having a mean gradient > 20 mmHg. The mean indexed AVA was 1.04 cm2/m2 for N-TAVR and

0.64 cm2/m2 for ViV-TAVR with severe patient prosthesis mismatch reported in 10% and 56% respectively.[1]

In 2021, **Abbass et al,** from the similar multicenter dataset recorded a discharge echocardiographic mean pressure gradient was 2x that measured immediately after valve deployment (10 vs 5 mmHg) and the percentage of patients with mean gradients > 20 mmHg increased from 2 (0.2%) to 30 (4%)[2] A major limitation of these reports is the inconsistency in data collection i.e. varying numbers with data at each time point.[1,2] In the older of the two reports, while not statistically analyzed, there were small reductions in AVA and iAVA from post deployment to discharge (1.04 to 0.9 cm2) and the incidence of severe PPM rose from 10% to 17%.2 Stroke volume index appeared to be higher at discharge compared to post deployment (31 to 37 ml/m2).[2]

Although data collection was inconsistent, these multicenter studies demonstrated discordance between echocardiographic and invasive transvalvular mean gradients after TAVR, which increased over the short period of time from valve deployment to the time of discharge.[1,2] If it is assumed that the invasive assessment is the reference method then the discordance suggests that echocardiography, which is currently the preferred method of follow-up, may not be adequate to determine prosthetic valve function.

Doppler derived gradients have limitations. The accuracy of Doppler the angle of interrogation. The Doppler Shift equation seen below involves cosine . The Doppler beam should be as parallel as possible otherwise the velocity measurement will underestimate the true flow velocity. Any misalignment between the Doppler beam and blood flow is likely to result in an underestimation of flow velocities.[3]

$$f = \frac{2v\, f_0 \cos\theta}{c}$$

$\cos 0° = 1$

Therefore, that echocardiography overestimates invasive assessment is surprising and concerning The estimated gradient is calculated using the Bernoulli equation:

$$\Delta P = \tfrac{1}{2}\rho(v_2^2 - v_1^2) + \rho(dv/dt)dx + R(v)$$

Convection Acceleration + Flow Acceleration + Viscous Friction

The majority of the time a simplified version is utilized for several reasons:[3,4]

1. Peak flows are of interest in clinical measurements. During peak flow, the flow acceleration is virtually nonexistent and thus can be ignored.
2. Viscous friction contributes significantly only in discrete orifices with an area of less than 0.25 cm2. Blood flow is thought to be constant for orifices with an area greater than this, so that viscous friction is also eliminated in the Bernoulli calculation.

$$\Delta PG_{max} = 4(v_2^2{}_{max} - v_1^2{}_{proximal})$$

3. For clinically significant lesions, v2 is substantially greater than v1 and v1 is typically < 1m/s, such that v22 – v12 is approximated by just v22.

$$\Delta PG_{max} = 4v_2^2{}_{max}$$

The data presented by Abbass et al reports that echocardiography derived gradients were higher than invasively measured data suggesting a need for additional considerations.[1] One consideration is the omission of the proximal or subvalve velocity (v1). When v1 is significant, i.e., > 1.5m/s, then it should be included in the pressure gradient equation. Baumgartner et al also states that if should be included when the peak velocity is < 3.0 m/s.4 Arguably in these conditions v2 is not significantly greater than v1.

$$\Delta PG_{max} = 4(v_2^2{}_{max} - v_1^2{}_{proximal})$$

Flow velocities across the outflow tract and valve may be lower in the presence of reduced ejection fraction, significant mitral regurgitation, and reduced ventricular preload/cardiac output. Another consideration is the effect of pressure recovery.[3,5,6] When flow velocity declines downstream from the aortic valve, kinetic energy is converted to potential energy, and there is pressure recovery. Inclusion would yield a smaller ΔPG.

$$\Delta PG_{net} = PG_{max} - \{PG_{max} \times 2 \times (EOA \times Ao_A) \times (1 - [EOA/Ao_A])\}$$

Including the subvalve velocity and considering pressure recovery would yield a smaller transvalvular pressure gradient. These were considered, by the same author, in an earlier smaller single center data set.7 Although the echocardiography derived pressure gradient was lower, it was still, on average, significantly greater than the invasively derived gradient.[7]

Other limitations in these studies and any echocardiographic measures of LVOT stroke volume and aortic valve area is the use of the two-dimensional assessment of LVOT (subvalve) diameter for the calculation of area.[1,2] Data has demonstrated has shown that this region is not circular in the majority of cases and that the diameter used in these calculations may represent the smaller dimension.[8] Without 3D assessment of area, it is more likely that these measures underestimate true SV and AVA.

Additional considerations include the variability of orifice shape and asymmetries. Depending on the presence of irregularities and the direction of the ultrasound beam, measured flow velocities and subsequently calculated pressures gradients will vary.[9,10] These are difficult to consider during routine echocardiographic imaging.

In the data presented by Abbass et al, the absolute discordance, on average, was not in the range of confusing normal and abnormal prosthetic valve function. Although echocardiographic gradients were higher than invasive data at the time of valve deployment and increased from the time of deployment to the time of discharge, neither absolute gradient, nor gradients > 10 or > 20 mmHg were associated with 30 day or 2-year mortality.[1,2] Instead, surgical risk predictors (STS) and an ejection fraction < 50% were predictors of 2-year mortality.[2]

Echocardiographically determined mean transvalvular gradients are relatively simple to obtain. While the mean echocardiographic gradients did not impact on mortality, the discordance still questions the reliability of echocardiography to accurately determine prosthetic valve function during follow-up. If, however, subvalvular velocities, pressure recovery, and use of 3D assessments were routine, then accuracy of valve assessment would improve, but certainly complicate the follow-up assessments and, the greater amount of data would likely introduce more variability and error among multiple sonographers. Perhaps the Doppler velocity index (or velocity ratio) might be a simpler, reproducible, and reliable assessment of prosthetic valve function that is independent of errors of two-dimensional measures and less reliant on actual flow.[11,12,13]

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Cardiac Remodeling in Subclinical Hypertrophic Cardiomyopathy: The VANISH Randomized Clinical Trial

Vissing CR, Axelsson Raja A, Day SM, Russell MW, Zahka K, Lever HM, Pereira AC, Colan SD, Margossian R, Murphy AM, Canter C, Bach RG, Wheeler MT, Rossano JW, Owens AT, Benson L, Mestroni L, Taylor MRG, Patel AR, Wilmot I, Thrush P, Soslow JH, Becker JR, Seidman CE, Lakdawala NK, Cirino AL, McMurray JJV, MacRae CA, Solomon SD, Bundgaard H, Orav EJ, Ho CY; Valsartan for Attenuating Disease Evolution in Early Sarcomeric Hypertrophic Cardiomyopathy (VANISH) Investigators. Cardiac Remodeling in Subclinical Hypertrophic Cardiomyopathy: The VANISH Randomized Clinical Trial. JAMA Cardiol. 2023 Nov 1; 8(11):1083-1088. doi: 10.1001/jamacardio.2023.2808. PMID: 37672268; PMCID: PMC10483382.

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Background

Hypertrophic cardiomyopathy (HCM) is an inherited cardiac disease with a general prevalence of 1:200-1:500.[1] HCM phenotypes encompass a spectrum of morphologies with unexplained left ventricular hypertrophy (LVH) as its defining feature. Left ventricular wall thickness (LVWT) can range from less than 15 mm in mild cases to greater than 30 mm in massive hypertrophy.1 The phenotypic variations in the morphology and severity of hypertrophy can be linked to different mutations of the genes encoding the cardiac sarcomere. Despite substantial improvement in treatment options for this patient population, which has considerable morbidity and mortality due to arrhythmias, heart failure, and sudden cardiac death,[2] the factors responsible for disease progression in early disease have yet to be defined. Previously, the Valsartan for Attenuating Disease Evolution in Early Sarcomeric Hypertrophic Cardiomyopathy (VANISH) trial demonstrated potential for valsartan to attenuate cardiac remodeling in patients with early stage sarcomeric HCM.[3] In this study, the authors aimed to explore the potential effects of valsartan to mitigate short-term disease progression in a parallel VANISH cohort of patients with subclinical HCM in which they were sarcomere variant carriers without LVH.

Methods

The VANISH multicenter double blind randomized clinical trial was conducted between 2014-2019 across 17 sites in Brazil, Canada, Denmark, and the United States. The trial consisted of 2 parallel cohorts: the primary cohort and the exploratory cohort. The primary cohort consisted of participants ranging from 8 to 45 years old with early-stage clinical HCM (178 total). The exploratory cohort consisted of participants ranging from 10 to 25 years old with no formal diagnosis of HCM but with early phenotypic manifestations of HCM, including increased LVWT to diameter ratio, ECG abnormalities, or reduced E′ velocity (34 total). After a 2- to 6-week valsartan titration period, participants in the exploratory cohort were randomized (1:1) to valsartan or placebo and continued treatment for 2 years. Visits were conducted at baseline, 1 year, and 2-year marks.

The effects of valsartan were studied by assessing composite outcomes of 9 biological components of cardiac structure and functioning, including biomarkers (serum troponin T, and NT-proBNP), echocardiography measurements (maximal LVWT, E′ velocity, and S′ Velocity), and cardiac magnetic resonance (CMR) imaging measurements (LV volume, left atrial volume and LV mass). Secondary outcomes assessed normalized changes of these 9 individual components. The composite outcome Z score was compared between groups in a linear regression model. The Wald test was used to determine statistical significance. Phenotypic progression was also analyzed via linear regression. Participants developing LVH were compared to those who were not using a t-test to determine statistical significance.

Results

Of the 34 participants with subclinical HCM, 100% identified as white. The mean age was 16 years old. The participants were evenly randomized between placebo and valsartan. The valsartan group did display modestly higher mean LA volumes (placebo, 27 mL/m2 vs valsartan, 33 mL/m2) and detectable troponin T concentrations (placebo, 0% vs valsartan, 23%). In the exploratory cohort, no significant differences between the valsartan and placebo groups were found in phenotypic progression (-0.01;95% CI, −0.29 to 0.26; P = .92). Secondary analyses of individual components were similarly neutral. Valsartan was well tolerated amongst participants with minimal effects on blood pressure (change in mean arterial pressure, −5.0 mm Hg [95% CI, −12.5 to 2.4mm Hg] with valsartan compared with placebo) and no instances of hyperkalemia or kidney dysfunction. Power of the study was limited to 30% as revealed by post hoc analysis.

Cardiac remodeling metrics remained stable over the 2-year period with only a modest increase seen in LA volume index (LAVI; 3.5 mL/m2 [95% CI, 1.4- 6.0 mL/m2]; P= .002). Troponin T levels became detectable in a minority of participants (5 out of 30 with undetectable troponin at baseline). About 26% of those with subclinical HCM showed progression in LVWT with 18% developing clinically overt HCM. Those who developed LVH tended to have larger body size, higher LVWT to diameter ratio, higher diastolic blood pressure and larger LAVI and IVS thickness. Logistic regression indicated LAVI and IVS thickness predictors for developing LVH.

When comparing participants with subclinical HCM to those with early stage HCM, the subclinical HCM cohort showed less phenotypic progression over 2 years. There were no differences in progression of disease in participants treated with valsartan across both cohorts however, a higher rate of progression was observed in the placebo group of the early stage HCM cohort.

Discussion

Despite the comprehensive approach of the VANISH trial, it faced many challenges in demonstrating an effect of valsartan on phenotypic progression of subclinical HCM. This was primarily due to small sample size and minimal phenotypic progression witnessed amongst cohorts. Regarding the limited sample size, which rendered the study underpowered, this suggests a need for further study with a significant increase in participants (220 to 500 participants per post hoc findings) to appropriately detect potential treatment effects. Notably, the study observed slow phenotypic progression in subclinical HCM with an even lower rate of cardiac remodeling compared to early-stage HCM.

However, it is difficult to say if this finding was due to an unmeasured imbalance of the cohorts in some way or the sheer difference in sample size between the cohorts. With regards to generalizability, the lack of diversity limits the clinical significance of these findings even if appropriately powered. For example, Wells et al. explore the role of race with regards to differences in disease progression and referral patterns in patients with HCM. They found that black patients in the US are more commonly referred with an uncertain HCM diagnosis for reasons like competitive sports clearance and are less commonly referred for symptom management. Black patients also have less advanced heart failure symptoms on presentation, making a study with such inclusion factors as this one particularly concerning.[4] Future studies should strive for both a larger and a more diverse participant pool in an effort to make a more substantial clinical impact.

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Routine Extubation in the Operating Room After Isolated Coronary Artery Bypass

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Background

Patients who undergo cardiac surgery on cardiopulmonary bypass (CPB) have traditionally been extubated postoperatively in the intensive care unit (ICU). Demonstrated benefits of early extubation following cardiac surgery include decreased length of stay (LOS), decreased cost, and improved perioperative morbidity.[1] It is unknown if the benefits of early ICU extubation following cardiac surgery also apply to in-OR extubation. Multiple recent studies evaluating in-OR extubations of select cardiac surgery patients have shown no increased rate of reintubations, no difference in morality, shorter hospital LOS, shorter ICU LOS and lower costs compared to in-ICU extubations, although one study found a higher rate of reoperation for bleeding and postoperative reintubation.[2-4] Even so, relatively little data have been published regarding the clinical outcomes of cardiac surgery patients extubated in the OR following full sternotomy and CPB without a minimally invasive surgical approach. Given the possible benefits and still unclear outcomes associated with routine in-OR extubations of cardiac surgery patients, more research is needed on this topic.

The authors of this single-center, retrospective study examined perioperative morbidity and mortality of OR-extubated and ICU extubated patients who underwent non-emergency, isolated coronary artery bypass grafts (CABG) that required CPB.

Methods

The study took place at New York University (NYU) Langone Hospital and examined isolated CABG operations between January 1, 2017, and December 31, 2022. All patients who underwent non-emergent, isolated CABG were included in the study. Cases that involved combined valve-CABG operations, reoperation CABG, CABG with combined mechanical circulatory support (MCS) and emergency procedures were excluded from the study.

Over the first three years of the study, patients were primarily extubated in the ICU, but beginning in the fourth-year candidates deemed to be appropriate were extubated in the OR, according to an institutional change in policy. The decision for extubation was collaborative between anesthesiologist and surgeon, discussed prior to the case and again prior to extubation. Patients who had significant hemodynamic instability, significant inotropic support, MCS or high amounts of bleeding were not considered candidates. Anesthesiologists were instructed to limit narcotics, use tylenol for analgesia and propofol for sedation prior to OR extubation.

The primary outcome measured in the study was reintubation,within 30 postoperative days from initial extubation, including OR-extubated patients who required reintubation in the OR. Secondary outcomes measured were reoperation for bleeding, reoperation for any cause, total number of grafts performed, total operative time, time from skin closure to OR exit, prolonged mechanical ventilation, postoperative atrial fibrillation (POAF), postoperative stroke or transient ischemic attack (TIA), postoperative renal failure requiring hemodialysis, ICU hours, postoperative LOS, discharge disposition, 30-day readmission rate and 30-day mortality.

The authors used propensity score matching to adjust for confounders between OR extubated and ICU extubated patients, which included age, body mass index (BMI), ejection fraction (EF), Society of Thoracic Surgery (STS) Mortality risk score, STS prolonged ventilation time, CPB time and aortic cross clamp time.

Results

Over the period of study, 1397 patients met inclusion criteria, of which 891 (63.8%) were ICU-extubated and 506 (36.2%) were OR-extubated. The percentage of OR-extubated patients greatly increased in the fourth year of the study according to the change in clinical policy. Propensity matching of cohorts generated 414 pairs, with no differences between sex, race, incidence of IABP (preoperative or intraoperative) or any other major comorbidities.

Across propensity-matched pairs, no significant differences were found in incidence of reintubation at 30-days (1.7% vs 1.7%, p =1.00). Similarly, no significant differences were observed in rates of reoperations for bleeding (0.7% v. 1.7%, p= 0 .2036), mean total OR time (390 v 394 minutes, p = -0.3202), rates of postoperative stroke or TIA (0.5% v 0.5%, p =1.0), postoperative renal failure (0.5% v 0.2%, p =0.5653) or 30-day mortality (0.7% v 0.2%, p =0.3167). By contrast, ICU-extubated patients were found to have a significantly higher incidence of prolonged ventilation (3.6% v. 1.0%, p = .0106) and POAF (15.0% v. 5.1%, p <.0001), as well as significantly more ICU hours, longer postoperative LOS, higher 30-day readmission rate and higher rates of discharge to an extended care facility versus directly to home.

Across the complete cohort, comparisons of in-OR versus in-ICU extubations yielded significant or non-significant differences in the same categories as propensity-matched comparisons, with the exceptions of total number of grafts and total OR time; the latter were significantly greater in the ICU group in non-propensity-matched analyses only.

Discussion

This propensity matched cohort study demonstrated the safety of in-OR extubating of patients who underwent non-emergency, isolated CABG on CPB. OR-extubated patients not only showed no significant increase in reintubation rate within 30 days of the initial surgery (primary outcome), but also had better postoperative outcomes in certain categories despite matching baseline characteristics and comorbidities.

Limitations of Study

This study is subject to several limitations. In addition to being retrospective, the study was not an intention-to-treat analysis. Therefore, patients who were originally planned for in-OR extubation could "crossover" to in-ICU extubation pending anesthesiologist and surgeon judgment. The authors did not collect data on how often such a situation happened. These crossovers may have biased the results. In addition, propensity score matching cannot account for unmeasured variables affecting how the anesthesiologist and surgeon determined which patients to extubate in the OR vs ICU. Finally, the patients in this study had a generally low risk profile, demonstrated by an STS mortality risk score of 0.8%, which limits the applicability of the results to higher risk patients.

Conclusion

The results of this study suggest that in-OR extubations following uncomplicated cardiac surgery, such as non-emergent CABG, is feasible and possibly beneficial compared to ICU extubations.

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Lead Extraction and Mortality Among Patients with Cardiac Implanted Electronic Device Infection

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Background

As the indications for cardiac implantable electronic devices (CIEDs) are expanding, the number of implantations has increased, with 300,000 implantations occurring only in the United States annually.[1-3] Despite significant efforts to decrease complications, the incidence of potentially lethal device related infection and endocarditis increases over time. In one study,[4] 2.4% rate of device related infection was found in 3 years after implantation while in another study,[3] 6.2% and 11.7% infection rate was found at 15 and 25 years, respectively. Because of the high mortality of CIED related infection if treated conservatively with antibiotics only,[5-7] early and complete device removal is recommended by guidelines and consensus statements as a class I intervention.[7-9] In a recent study[10] of 25,303 admissions for CIED infection/ endocarditis between 2016 and 2019, only 11.5% were treated with device extraction, implying possibly nonadherence to the recommendations. The odds of death were lower among patients who had compete CIED extraction.

In the present study, the authors analyze the outcomes of Medicare patients with CIED infections in a nationwide clinical practice cohort.

Methods

In this cohort the data were from Medicare fee-for-service (FFS) between 1/1/2004 and 12/31/2019. The study was approved by the Duke University IRB for data assessment through Centers of Medicare & Medicaid Services Chronic Conditions Warehouse Virtual Research Data Center.[11] Study population consisted of patients with de novo insertion of AICD or pacemaker (N= 1 065 549). Patients with diagnosis of CIED infection or endocarditis, provided they received antibiotics within 30 days from diagnosis were included. Those with infection within 12 months of device implantation were excluded as these infections are usually superficial or minor wound healing issues.

Additionally, patients with device removal within 30 days prior to the diagnosis of infection were also excluded, as this was a delayed diagnosis. Final cohort of CIED infection n= 11 304 patients were further categorized into male, female, black, white, other and regarding the timing of device extraction, as follows:

(1) no extraction within 30 days from infection n= 9202
(2) extraction within 7 days from infection n= 1511
(3) extraction at 7-30 days from the diagnosis of infection n= 591

The primary outcomes were device infection, extraction, and all-cause mortality.

Statistical analyses were performed using SAS Enterprise Guide version 7.15.

Results

Mean (SD) follow-up from device implantation: 4.6 (2.9) years.

Patients with CIED:	1 065 549	Patients with CIED infection: 11 304
(1.1% mean age (years):	78 (72-84)	75 (67-82)
Male%/Female%:	50.9/ 49.1	60.1/39.9
Black%/White%	Other%: 7.6/ 87.2/ 5.2	15.7/ 77.2/ 7.2

The patients who developed CIED infection were sicker: they were more likely to have

comorbidities such as diabetes, COPD, kidney disease, CAD, heart failure and PVD.

Patients who received a defibrillator, with or without resynchronization therapy (CRTD or ICD), were more likely to develop infection in comparison with the patients who received a pacemaker with (CRTP) or without resynchronization therapy.

The incidence of CIED infection increased over time. The one-year survival after diagnosis of CIED infection was 68%. Only 18.6% (2102) of the patients diagnosed with CIED infection underwent extraction within the first 30 days, of which 13.4% (1511) within the first 6 days and 5.2% (591) on days 7-30.

At 5 years the incidence of extraction reached 25.4% (2787). Female, Black, and other race patients were less likely to undergo device/lead extraction within 30 days from diagnosis of infection, compared to Male and White patients. Sicker, frail patients with diabetes, TIA, CVA or with kidney disease were less likely to undergo extraction within 30 days from infection. Patients with an AICD were more likely to undergo extraction within 30 days from infection compared to patients with a pacemaker. Among patients with endocarditis, only 16.7% had extraction within a year after diagnosis.

The observed incidence of extraction increased over time: 15.6% in 2007, 20% in 2012 and 24.8% in 2019.

One year Mortality
No device/ lead extraction: 32.5%

Device /lead extraction on days 7-30 after the diagnosis of CIED infection: 23.4% vs 18.6% when extraction was performed within the first 6 days from diagnosis of infection.

Among patients with endocarditis, one year mortality without extraction was 31.4% vs 24.9% and 27.7 when extraction occurred the first 6 days or between days 7 and 30, respectively.

From multivariate analysis: extraction was associated with lower mortality than non-extraction. Female sex and older age were associated with higher mortality.

Discussion
In this large nationwide clinical practice cohort, only approximately a fifth of the patients with device or lead infection or endocarditis underwent device and lead removal. High mortality associated with non-removal or with delayed removal was also demonstrated.

Because complete hardware removal in the presence of infection is a class I recommendation[7-9] and prior studies have shown that:

1. Perioperative mortality of lead extraction is 0.3-0.5% [12,13]
2. There is approximately 50-100% recurrence of the infection without complete removal of the hardware [14-17]

Stricter adherence to the guidelines and earlier device extraction in the presence of infection will improve outcomes.

Limitations of the Study Include:
Because this study is a retrospective analysis of Medicare Claims, incomplete or inaccurate coding can affect the accuracy of the results.

Information such as whether the patients were pacemaker dependent or the BMI or the microbiologic cause of infection and bacteremia which can play a role in outcomes was unknown.

The strict exclusion criteria and study of specific scenarios, excluding other scenarios such as device reimplantation, may have affected the results.

The study population, Medicare patients, is a high risk for events population and decisions may be made with other parameters in mind compared to the general population.

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PRO CON

PRO/CON DEBATE

INTRODUCTION:

Inotrope Selection in the Presence of Pulmonary Hypertension and Right Ventricular Dysfunction

Acute pulmonary hypertension (PHTN) with right ventricular (RV) failure is associated with up to 30-40% in-hospital mortality.[1] More than 50% of PHTN is due to left heart (type 2) or pulmonary (Type 3) dysfunctions, followed by pulmonary arteriopathy (Type 1) and chronic thromboembolic disease (Type 4). Although treatment is directed at the primary dysfunction, management of acute PHTN, RV failure with hemodynamic dysfunction includes two general principles:[2]

1. **Establishment of right ventricular (RV) — pulmonary artery (PA) coupling**
2. **Optimization of ventricular interdependence**

Right ventricular–pulmonary artery coupling refers to a relationship between the RV and the pulmonary artery that reflects on the RV's ability to overcome pulmonary vascular resistance (PVR; RV afterload).[2] Under normal conditions, the amount of energy needed for blood to flow from the RV to the PA is minimal and consistent with the RV pressure-volume curve with ill-defined or even absent isovolumic periods, suggesting that the RV is a passive conduit for blood to pass into the pulmonary arterial tree.[3–9]

The idea that blood can pass into the pulmonary system without a contracting RV has been demonstrated experimentally and clinically.[10–14] Although normal RV contractility is preferred, it is not necessary to maintain hemodynamic stability if there is a pressure gradient to drive blood forward from the RV. Normally, and especially in the presence of RVSD, the left heart contributes significantly toward RV systolic performance, i.e. pressure generation (dP/dt) and pulmonary blood flow (Qpa).[8,15–17]

Ventricular interdependence is largely driven by the interventricular septum (IVS), which is normally concave toward the RV in diastole and becomes more so during ventricular systole.[18,19] The position and shape of the IVS depend on pressure gradients between the left ventricle (LV) and RV. Coupled with connectivity and contraction of the left and right ventricular free wall fibers, the LV may be responsible for nearly 70% of the RV dP/dt and Qpa. For this to happen, the RV and LV are anatomically related such that the RV appears wrapped or stretched around the more symmetrical bullet-shaped LV.[18,19]

In this light, the management of RVSD and PHTN involves a comprehensive and interdependent interaction of preload, afterload, contractility, heart rate, and rhythm. Preload optimization seeks to maintain normal anatomic relations between the left and right heart, to allow proper ventricular interdependence.[20–23] Excess right heart volume causes left septal shift during diastole, compromising left heart filling, performance, pressure generation, and cardiac output.[20–26]

The position of the IVS is also related to the relative right and left-sided pressures, represented by either mean and systolic systemic pressure (MAP; SAP), mean and systolic pulmonary artery pressures (mPAP; PASP), or respective pulmonary and systemic vascular resistances (PVR, SVR; PVR/SVR).[27–29] A reduction of RV afterload (mPAP, PASP, PVR; Ea) is an important target in managing patients with RVSD and PTHN and can be partly reflected by PVR/SVR ratio (or mPAP/MAP or PASP/SAP). Maintaining or elevating systemic pressures not only improves septal position and shape but is important to improve right heart coronary flow to match the increased myocardial oxygen consumption.[24–26]

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Improving ventricular contractility and optimizing ventricular interdependence improves forward flow (QPa). Even if RV contractility does not improve acutely, improving LV contractility, perhaps specifically the IVS, improves global RV performance (RV Ees).[7,28] It is possible that RV Ees can occur without an improvement in RV myocardial contractility. By optimizing preload and anatomic relations, systolic ventricular interdependence is magnified, improving RV-PA coupling and generating RV dP/dt and QPa.[8,20,23]

Finally, maintenance of atrial-ventricular synchrony and avoidance of tachy- or bradyarrhythmias is important to maintain cardiac loading conditions and forward flow. While bradycardias cause chamber dilation, strain, and dysfunction, tachyarrhythmias and AV dyssynchrony and impair filling, forward flow, and coronary filling time.[24,30,31] In the presence of RV failure, there is a dependency on the sympathetic neural input.[30] Higher normal heart rates, perhaps mild sinus tachycardia, reduce chamber dilation and pressures, and improve cardiac output and forward flow.[24,31]

In considering the ideal inotrope for the patient with pulmonary hypertension and right heart failure, it is necessary to consider how all the variables interact. Whether or not right heart failure is secondary and represents the severity of a primary dysfunction, or is the primary of causing cardiovascular failure, the associative outcomes are directly to right heart dysfunction. While management is directed toward the primary causative etiology, addressing right heart dysfunction, and restoring ventricular interdependence and RV-PA coupling is necessary to stabilize the patient i.e., addressing PHTN and RVSD.

The following Pro explores and uses of Dobutamine, Milrinone and Dopamine as the first line inotropic medication for right heart failure.

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Effects on the cardiovascular and pulmonary systems

	α_1	β_1	β_2	DA	Inotropy	HR	MAP	PVR	SVR	SVR/PVR	RV Ees/Ea	Misc
Dobutamine	↑	↑↑↑↑	↑↑↑	NC	↑↑↑	↑↑↑	Variable	↓	↓	↑/NC	↑↑	↑HR;↓BP Arrhythmia
Epinephrine	↑↑↑	↑↑↑	↑↑↑	NC	↑↑↑	↑↑↑	↑↑↑	↑	↑↑↑	↑	NC/↑	↑HR; Arrhythmia Lactic Acidosis; Hyperglycemia
Milrinone	0	0	0	0	↑↑↑	↑↑↑	↓↓	↓↓	↓↓	↓	NC/↑	↑HR;↓BP Arrhythmia
Dopamine (1-4 ug/kg/min)	NC	↑	NC	↑↑↑↑	↑↑	NC	↓	↓	↓	NC	?	↓BP
Dopamine (5-10 ug/kg/	↑	↑↑↑	NC	↑↑↑↑	↑↑↑	↑↑↑	↑↑↑	NC	↑↑	↑	?	↑HR; Arrhythmia
Dopamine (10+ ug/kg/min	↑↑↑	↑↑↑	NC	↑↑↑↑	↑↑↑	↑↑↑	↑↑↑	Variable	↑↑↑	↑	?	↑SVR,↑ PVR,↑mPAP

DA = Dopamine; HR = Heart Rate; MAP = Mean Arterial Pressure; PVR = Pulmonary Vascular Resistance; SVR = Systemic Vascular Resistance; RV Ees/Ea = Right Ventricular-Pulmonary Artery Coupling

Dobutamine is the Preferred Inotrope for Patients with Pulmonary Hypertension and Right Ventricular Dysfunction

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The management of right ventricular (RV) dysfunction centers around optimizing preload, afterload, and contractility. In patients with RV dysfunction due to left ventricular (LV) dysfunction, optimizing the left heart may improve RV function. RV function also requires adequate interventricular septal wall function, which may account for up to 40% of RV systolic function.[1]

Optimizing RV afterload involves managing pulmonary arterial hypertension (PAH). PAH can result from several causes. World Health Organization (WHO) Group II PAH results from LV dysfunction, and treatments are focused on improving LV function.[2] RV systolic dysfunction in the setting of non-Group II PAH can be challenging to treat, with clinical practice variability in inotrope choice.[3,4] While it may be tempting to extrapolate therapeutic strategies employed in LV dysfunction management, such an approach may yield unfavorable outcomes. Within this clinical equipoise, we posit that dobutamine is the ideal inotrope in the setting of RV dysfunction and PAH.

The RV is a complex, conical structure as compared to the elliptical LV. Consequently, the imaging, volume quantification, and functional assessment can be more complex with no one measurement having as lasting a clinical permanence as LV ejection fraction. Maladaptive RV changes include RV hypertrophy and dilation, which add complexity in quantitative longitudinal echocardiographic assessment. RV chamber dilation can lead to functional tricuspid regurgitation, which can worsen cardiac output and exacerbate organ venous congestion in a vicious cycle.[5]

The RV and the pulmonary artery (PA) are connected in series. To examine the system of the RV-PA unit, pulmonologists, physiologists, and cardiopulmonary experts utilize values to define RV contractility (ventricular elastance (Ees)) relative more clearly to its afterload (arterial elastance (Ea)). The usefulness of Ees as a measure of RV contractility is, for example, seen in disease states where global RV function is reduced, but Ees is increased to match an appropriate pulmonary afterload. The RV-PA unit in this scenario is said to be "coupled." When there is appropriate RV-PA coupling, the adapted contractility of the RV matches PAH.6 Although the specifics of these measurement techniques are beyond the scope of this argument, the concept of RV-PA coupling is relevant to the use of dobutamine to augment RV contractility without a precipitous increase in pulmonary vascular resistance (PVR).

Dobutamine, derived by modifying the chemical structure of isoproterenol, is a primarily beta-1 adrenergic receptor agonist, and it augments cardiac contractility. There is also some beta-2 agonism, which reduces systemic vascular resistance, and a small amount of alpha-1 adrenergic agonism.[7] In a porcine study, dobutamine demonstrated improvements in RV-PA coupling and RV function through increased RV contractility and decreased PVR.[8] Additionally, the improvement in RV-PA coupling using dobutamine enhances RV ejection in heart failure patients with preserved ejection fraction subjects through afterload reduction alone, indicating dysfunction in RV systolic reserve rather than enhanced contractility.[9]

The other commonly used inotrope in this clinical scenario is epinephrine, which is a sympathomimetic catecholamine with alpha-1, alpha-2, beta-1, and beta-2 agonism. Lower doses have a primarily beta receptor effect, while higher doses increase SVR through alpha-receptor agonism. There are no randomized controlled trials comparing dobutamine and epinephrine in this clinical scenario, and there is significant practice variation by institution and provider.[4] Numerous studies have documented the underwhelming

efficacy of epinephrine in states of mixed shock, with some reporting up to a three-fold increase in mortality in cardiogenic shock, potentially attributed to alterations in cardiac microcirculation and calcium homeostasis.[10] Moreover, in contrast to the use of other vasopressors, epinephrine is linked to a significant exacerbation of cardiac stress, myocardial injury, and kidney dysfunction within the four days following shock detection.11 The drawbacks associated with epinephrine use in cardiogenic shock encompass excessive lactic acid production, tachycardia, heightened myocardial oxygen demand, increased arrhythmias, and multi-organ toxicity.[10] Additionally, at high doses, epinephrine directly increases PVR, requiring increased RV contractility to maintain RV-PA coupling, if there is RV contractile reserve.[12]

When compared to milrinone, Dobutamine was shown to be a more complete inotrope.[13]

After identifying patients with severe congestive heart failure, half were randomized to receive intravenous milrinone (50 micrograms/kg bolus then 0.5 microgram/kg/min) and half received dobutamine (2.5 to 15 micrograms/kg/min) to achieve equal increases in cardiac output. Both drugs significantly improved cardiac performance, similar increases in RV ejection fraction, nearly identical increases in mean cardiac index and no change in heart rate. Right atrial pressure and right ventricular volumes were unchanged. However, the mechanism of improvement was different. While milrinone's mechanism was more or less a reduction in RV afterload reduction, dobutamine's effect was inotropic augmentation.[13]

The management of patients with RV dysfunction and PAH necessitates tailored therapy. The care of these patients is incredibly nuanced and requires a multidisciplinary approach with the use of multiple agents, including inhaled pulmonary vasodilators, volume optimization, and ventilation optimization. There are also emerging biologic therapies of unknown significance in this vulnerable population.[14] There is also a significant consideration for the use of mechanical circulatory support.[15] If choosing chemical RV support in the setting of PAH, the current evidence suggests using dobutamine.

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Milrinone — The Agent of Choice for Right Ventricular Dysfunction and Pulmonary Hypertension

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Inotropic agents increase the force of cardiac muscle contractions. For reasons including chronic dysfunction, acute changes (e.g., afterload mismatch), and the effects of cardioplegia, the need for inotropic support to facilitate separation from cardiopulmonary bypass (CPB) is significant. The presence of pulmonary hypertension and right heart dysfunction complicates management further.[1,2] Even in the patient without pre-existing pulmonary hypertension, acute changes due to pulmonary dysfunction can be significant and cause right ventricular dysfunction.[3] The management and selection of inotropic agents for patients with pulmonary hypertension (PHTN) depends on the primary etiology, the presence of right ventricular strain and whether or not the pulmonary vascular resistance (PVR) is elevated or not.[4,5] While PHTN can be divided based on the PVR, patients with PHTN, regardless of type, is associated thickening and abnormal architecture of the pulmonary vascular wall.[6] For cardiac surgical patients, the ease of weaning from cardiopulmonary bypass (CPB) is directly related to the ability to maintain or increase the ratio between the mean arterial pressure (MAP) and mean pulmonary artery pressure (mPAP).[7] Combining inotropy and right ventricular afterload reduction is optimal to restore coupling between the RV and the pulmonary vasculature and ventricular interdependence with the left ventricle.[2,8,9]

Phosphodiesterase (PDE) III is found in cardiac myocytes and peripheral vascular smooth muscle cells and breaks down cAMP. PDE inhibitors (PDEi) impede the metabolism of secondary messengers; cyclic adenosine monophosphate (cAMP) and cyclic guanosine monophosphate (cGMP), thereby increasing their concentration in myocardial and vascular smooth muscle cells.[10] In myocytes, this increases calcium influx increasing myocardial contractility.[11] In pulmonary and systemic vascular smooth muscle cells, the accumulation of cAMP and cGMP activates PKA and cGMP-dependent protein kinase G (PKG) that cause potassium channel mediated hyperpolarization and vascular smooth muscle relaxation, i.e., systemic, and pulmonary vasodilation.[9,12] Although the benefits of PDEi for heart failure is well documented, their use may be especially useful for the acute and acute on chronic heart failure patient in which adrenergic receptors are downregulated or desensitized.[9,13]

Milrinone is a phosphodiesterase (PDE) III inhibitor with positive inotropic and lusitropic properties that also promotes vasodilation of the systemic and pulmonary vasculature.[4,9,10] Considering these benefits, Milrinone is the ideal drug for managing heart failure with pulmonary hypertension and right ventricular/heart dysfunction.[9,14]

Current literature supports the prophylactic administration of milrinone to facilitate weaning and separation from cardiopulmonary bypass (CPB), perhaps even more so for patients with right ventricular (RV) dysfunction.[2,9,14,15] Even if its inotropic effects increase myocardial oxygen consumption, the afterload reduction counters it so that myocardial oxygen consumption is not increased.[16] Milrinone's benefits in patients with pulmonary hypertension and right heart dysfunction are demonstrated for both intravenous and inhalation routes.[1,15,17,18] Administration by inhalation is relatively selective for the pulmonary vasculature with little effect on the systemic circulation.[1,18]

Hemodynamically, milrinone increases cardiac index, reduces, or maintains systemic vascular resistance, and reduces especially pulmonary artery and right heart pressures.[19] For patients with responsive vasculature, milrinone lowers pulmonary vascular resistance.[19] The main adverse effects include hypotension and dysrhythmias especially in patients with renal impairment.[20] Typically, milrinone has a positive chronotropic effect but this is not consistent

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and occasional bradycardia may occur.[14] Systemic vasodilation is known to occur with Milrinone possibly requiring co-administration of a vasopressor.[14,21,22]
For patients with increased right heart strain and pressures, maintaining coronary flow is important to preventing further deterioration. Contrasting catecholamines with -1 vascular constriction, Milrinone is reported to vasodilate the internal mammary artery, and was superior to nitroglycerin in patients receiving -1 medications to improving coronary flow.[14,23] In addition to improving internal mammary artery flow, Milrinone significantly increases the flow in anastomosed saphenous vein grafts after CPB.[24] Considering that Milrinone is an inotrope that reduces pulmonary artery pressures and vascular resistance, it is the best inotrope for patients who have PHTN and right heart dysfunction.[18]

Clinically, milrinone increases the likelihood of a successful first wean from cardiopulmonary bypass (CPB) without increasing catecholamine requirement but often with additional vasopressor support.[13,14,15,18,21,22,25,26] Milrinone has known benefits for patients with pulmonary hypertension.[2,8,27] In many centers, milrinone is considered the drug of choice for managing patients with right ventricular (RV) failure or high pulmonary artery pressures complicating separation from CPB.[15] In patients with chronic pulmonary hypertension, milrinone improved RV contractility, reduced afterload, and increased pulmonary blood flow.[27]

In a neonatal piglet shock model with hypoxic PHTN, Milrinone prevented continued increases in pulmonary vascular resistances after hypoxia-reoxygenation.[16] In a pig model of pulmonary regurgitation with cardiac failure, right ventricular dilation and dysfunction and a leftward shift of the interventricular septum, Milrinone infusion significantly increased RV contractility, cardiac index, and mixed venous oximetry while reducing central venous pressure.[17] Aside from its hemodynamic benefits, Milrinone improve oxygenation.[19,22]

Although milrinone is most commonly administered intravenously, inhaled milrinone has received interest as a selective pulmonary vasodilator administered via a common nebulizer.[15,28,29,30] Compared to intravenous administration, inhaled milrinone reduces pulmonary artery pressures and pulmonary vascular resistance with minimal systemic vasodilation.[15,28,29,30] Prophylactic administration of inhaled Milrinone has been reported to improve facilitate separation from CPB and reduce the amount inotropes needed in the postoperative period.[15,18,31] When given prophylactically in high-risk cardiac surgical patients prior to initiation of bypass inhaled milrinone lowered pulmonary artery pressure after bypass and was associated with fewer cases needing emergency re-initiation of bypass after weaning.[21] Gebhard et al provides support for intratracheal administration of milrinone to facilitate CPB weaning in patients with acute RV dysfunction, with less additional vasopressor need in the early postoperative period.[15] Inhaled milrinone is also useful for the treatment of severe pulmonary hypertension and acute lung injury. In a large RCT, milrinone significantly reduced the incidence of low cardiac output syndrome in a neonatal and pediatric population.[32]

Considering that Milrinone is an inotrope that reduces pulmonary artery pressures and vascular resistance, it is the best inotrope for patients who have PHTN and right heart dysfunction.[2,8,18] It is evident that the inodilator properties of Milrinone improve right ventricular-pulmonary artery coupling with a return toward a more normal interactive state to improve forward blood flow. More recent metanalysis comparing patients with acute decompensated heart failure and cardiogenic shock reported a benefit with the use of milrinone compared to dobutamine.[33] In 11 patients with severe heart failure, both milrinone and dobutamine increased cardiac index, however milrinone reduced left ventricular end diastolic pressure significantly more.[34] By comparison, Dobutamine increased myocardial oxygen consumption while milrinone reduced it.[34] In patients with mitral regurgitation and pulmonary hypertension, PDEi (enoximone) was equal to Dobutamine combined with nitroglycerin in increasing cardiac output, however, mPAP and the alveolar-arterial oxygen gradient were significantly lower with PDEi.[35]

The use of epinephrine in patients with pulmonary hypertension is generally approached

with caution and is reserved for specific situations.[9] Epinephrine is a hormone and medication that stimulates both alpha- and beta-adrenergic receptors. While it can be beneficial in certain scenarios, it also has potential risks, especially in the context of pulmonary hypertension.[9] Epinephrine can cause vasoconstriction, increasing pulmonary vascular resistance leading to increased workload on the right side of the heart, which can be problematic in pulmonary hypertension.[9]

For patients with acute on chronic RVSD and failing catecholamine therapy due to receptor downregulation, treatment with phosphodiesterase inhibitors may still be effective since their action is not receptor mediated. Inhibition of the phosphodiesterase enzyme in vascular smooth muscle leads to vasodilatation.[4] Data also demonstrate that Milrinone in combination with low dose catecholamine may further facilitate weaning from cardiopulmonary bypass and/or states of cardiogenic shock.[25,34]

Given milrinone's favorable hemodynamic profile in patients with acute RV dysfunction as well as it is pulmonary vasodilatory abilities, in both the adult and pediatric population, it is the ideal inodilator to use in patients with pulmonary hypertension secondary to left sided cardiac dysfunction.

For patients with increased right heart strain and pressures, maintaining coronary flow is important to preventing further deterioration. Contrasting catecholamines with -1 vascular constriction, Milrinone is reported to vasodilate the internal mammary artery, and was superior to nitroglycerin in patients receiving -1 medications to improving coronary flow.[14,23] In addition to improving internal mammary artery flow, Milrinone significantly increases the flow in anastomosed saphenous vein grafts after CPB.[24] Considering that Milrinone is an inotrope that reduces pulmonary artery pressures and vascular resistance, it is the best inotrope for patients who have PHTN and right heart dysfunction.[18]

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DOPAMINE: The Total Inotrope

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When considering these management principles of the failing RV with (or without) pulmonary hypertension, Dopamine may be the ideal inotrope. Dopamine is a natural catecholamine and the precursor of norepinephrine and epinephrine (1,2). Like other catecholamines, Dopamine receptors are coupled with G-proteins and activate adenylyl cyclase to increase cAMP and protein kinase-A activity, resulting in an influx of cellular calcium and myocardial contraction. In addition to stimulating , and -adrenergic receptors, Dopamine also stimulates Dopaminergic receptors (isoforms D1 through D5), perhaps more significantly D1 and D2 receptors (1,2). Dopamine's action on the cardiovascular system includes initiation and increase of heartbeat and contractility, which results in increased cardiac index, heart rate, stroke volume, and systemic blood pressure (1)

Hemodynamics

The pharmacologic effects of Dopamine have traditionally been taught to be dose-dependent; however, clinical variability exists. Although Dopamine is considered a vasoconstrictor and inotropic medication, lower doses, up to 5 ug/kg/min causes peripheral and central vasculature vasodilation through D1 and D2 receptors.[1,3,4] Low doses of Dopamine (0.5 - 5 mcg/kg/min) reduce renal, mesenteric, and variably peripheral arteriolar tone.[1,5-8] Renally, Dopamine increases electrolyte excretion and diuresis.[5-8] Although there is no conclusive data that Dopamine improves renal outcomes, it is evident that it promotes diuresis.[5-8] D1 receptor activity causes relaxation of the splanchnic bed, which harbors a large portion of the venous blood volume.[9,10] In total, Dopaminergic receptor stimulation may reduce cardiac preload, pulmonary pressures, and pulmonary vascular resistance.[9,10]

D2 receptors on the adrenal medulla have a tonic inhibitory effect of reducing epinephrine and norepinephrine concentrations in non-stress times.[9] However, during stress signaled by splanchnic nerve activity, D1 receptor stimulation results in catecholamine production and stimulation.[10] At an intermediate infusion rate (> 5 ug/kg/min), Dopamine directly stimulates and receptors. In total, Dopamine has a positive impact on preload reduction and cardiovascular stimulation, making it an excellent heart failure medication.[11] The overall effect on systemic pressures and systemic vascular resistance is mixed depending on infusion rates and stimulation of cardiac output.[12,13]

Intermediate doses of Dopamine (2 - 10 mcg/kg/min) have a positive inotropic and chronotropic effect through Dopaminergic and beta-adrenergic receptor activation, resulting in increased contractility, cardiac index, heart rate, stroke volume, and blood pressure.[1] Higher doses (> 10 mcg/kg/min) have increasing vasopressor effects, increasing systemic vascular resistance through stimulation of alpha-adrenergic receptors.[2] There is also evidence that stimulation of alpha receptors can have a positive inotropic effect in both the right and left ventricles.[14]

Several animal model studies have corroborated Dopamine's hemodynamic effects. Wider ranges of Dopamine doses than those used commonly in humans have demonstrated that at low doses (< 5ug/kg/min), there is a decline in MAP and SVR; and at higher doses (80ug/kg/min), there is an increase in SVR and MAP, and insignificant changes in mPAP, and no change in PVR.[4] These studies show no significant changes in PAP or PVR, while systemic pressures and resistance increase along with cardiac output.[15,16] In another study involving Dopamine infusion rates up to 320 ug/kg/min, MAP and SVR increased from 5-320 ug/kg/min, PAP did not increase until 40 ug/kg/min, and CVP declined throughout the entire dosing range.[16] Changes in heart rate were not seen until a dose > 10ug/kg/min in one study and >160 ug/kg/min in another.[4,16] Overall, hemodynamic effects include increased cardiac output/index, stroke volume, blood pressure and SVR/PVR, with minimal changes in left atrial, pulmonary pressures and PVR, and a decline in central venous pressure.[4,15,16]

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The concerns regarding Dopamine causing cardiac ischemia are not supported. Dopamine administered at rates of 2-30 g/kg/min did not affect left heart pressure or heart rate significantly.[15] In an animal model using a wide infusion range of Dopamine infusion of up to 320 g/kg/min, the coronary vascular resistance decreased, and coronary flow increased linearly from 10-40 ug/kg/min and remained as such up to 320 ug/kg/min.[16] Left ventricular and pulmonary artery pressures did not increase until 40 ug/kg/min.[16] While myocardial work increased, coronary arterial-sinus oxygen content decreased.[16] Based on studies including dopaminergic, beta-, or alpha-adrenergic receptor blockade, authors concluded that direct or indirect vasodilator receptors (Dopaminergic, 1-, and 2-adrenoceptors) increased coronary blood flow.[17]

Non-Cardiac Benefits

Dopaminergic actions on noncardiac organs through D1 receptors result in vasodilation and increased flow to renal, mesenteric, carotid, and cerebral vascular beds.[18,19] When administered during cardiopulmonary bypass, peripheral flow to the splanchnic tissues increased in the exclusion of changes in cardiac function.[20,21] In an animal model, Dopamine infusion at 20 g/kg/min reduced mesenteric vascular resistance and increased mesenteric flow.[18] Improved splanchnic blood flow was demonstrated by higher gastric intramucosal pH due to selective vasodilatation of the splanchnic vessels.[22]

Peripheral organ flow is increased either by Dopaminergic receptor stimulation or increased perfusion pressure due to increased cardiac output.[17,22,23] In a meta-analysis, dopamine administration increased urine output by 24% over the first 1-2 days.[23] Dopamine, combined with mannitol and furosemide, administered postoperatively for cardiac surgical patients with renal failure, despite adequate cardiac output, increased diuresis, and reduced the need for renal replacement therapy.[24] Even if renal outcomes aren't improved, the diuretic effect of Dopamine may help manage preload in patients with right heart failure and congestion. This is supported by animal models reporting reductions in central venous pressure.[4,15,16]

Pulmonary Hypertension and Right Ventricular Failure

Managing PHTN and RVSD complicated by hemodynamic instability requires optimizing ventricular interdependence and improving RV-PA coupling. Realistically, management is not limited to one drug but includes multiple therapeutic angles, including optimizing RV preload, reducing RV afterload, improving RV contractility, maintaining, or increasing perfusion pressures and coronary flow.[25] Avoiding tachyarrhythmias and compromising bradycardia are important to optimize preload and output.[25] Is it possible to accomplish all this with only one drug? Perhaps not, but Dopamine comes closest.

Preload optimization can be described by the IVS position and shape, both being dictated by pressure gradients between the LV and RV and by relative volumes. Although the central venous pressure may not accurately reflect volume in the failing RV, venous hypertension (>12 mmHg) is known to compromise end-organ perfusion.[13] Dopamine is associated with diuresis and reduced central venous pressure.[16,26-28] Optimizing preload permits the rightward shift and concavity of the IVS helping facilitate ventricular interdependence. An overfilled high-pressure RV will minimize the impact of systolic ventricular interdependence.[12,13] From the ventricular short axis or four chamber windows, the short axis dimension of the RV is ideally 0.6 of the LV and hopefully < 1.0.[12,13] Maintaining lower right sided pressures and volumes as well as increasing SVR/PVR normalize the septal position and shape. As stated above, management may require additional considerations. Combining Dopamine with Nitroglycerin results in improved forward flow and lower filling pressures, including a reduced central venous pressure.[29,30]

Reduction in RV afterload, reflected by decreases in pulmonary vascular resistance and pressure improves RV-PA coupling and is critical for forward blood flow in the poorly contracting RV. The ability of the RV to generate a pressure exceeding that of the pulmonary circulation drives blood forward. Since recovery of RV contractility is not assured, systolic

ventricular interdependence is critical for the generation of RV dP/dt. The septal shape and position are related to the relative LV to RV pressure gradients, perhaps reflected by the SVR/PVR (MAP/mPAP) ratio.[12,31,32] Feltes et al reported very little change in mPAP and PVR, over a large infusion range of Dopamine (0-160 ug/kg/min), while MAP, SVR, and cardiac output were significantly increased.[4] A number of pulmonary hypertension models report the benefits of Dopamine infusions.[18,33-37] In piglets, two hours of hypoxemia followed by reoxygenation resulted in reduced MAP and increases in PVR, mPAP, and mPAP/MAP.[18] Both Dopamine (20 g/kg/min) and Dopamine (10 g/kg/min)/Epinephrine (0.1 g/kg/min) increased cardiac index and MAP. Dopamine alone resulted in a small reduction of mPAP/MAP (94% of pre-study baseline), while adding epinephrine resulted in mPAP/MAP, which was 101% of baseline.[18] In another animal study of pulmonary embolism which increased mPAP and decreased cardiac output, the infusion of Dopamine decreased PVR and increased cardiac output by 50%.[33] On balance, these studies describe an increase in the SVR/PVR ratio, which is beneficial toward the IVS position.

In a study of Dopamine's hemodynamic effects in patients with PHTN, Dopamine infusion increased heart rate (82 to 111 beats/min), mPAP (38 to 49 mmHg), MAP (85 to 98 mmHg), and cardiac index (2.0 to 3.0 L/min/m2).[38] Pulmonary vascular resistance did not change significantly (4.5 to 4.3 WU).[38] Six of the ten patients had significant reductions in PVR; three reported increases, and one appeared equivocal.[39] The authors concluded that the rise in mPAP was the result of increased forward flow to a non-compliant pulmonary arterial tree.[38] The authors further stated that Dopamine was 'an excellent agent for improving the circulatory state of patients with decreased ventricular function' for this population.[38]

Dopamine is effective at optimizing ventricular interdependence. Two right heart ischemic failure experiments by Goldstein et al. reported on the benefits of Dopamine.[34,35] In one study, atrioventricular pacing was varied to demonstrate the dependency of right ventricular performance on systolic ventricular interdependence and left ventricular function.[35] Ventricular interaction is driven by contraction and concavity of the IVS. By reducing the RV-free wall to IVS distance, the RV preload is compressed to generate RV dP/dt and pulmonary blood flow (QPa).[35] In a second study, right atrial and right ventricular free wall ischemia were induced, followed by interventricular septal ischemia.[34] Although right ventricular free wall ischemia resulted in dysfunction and a slight increase in right atrial pressure, systemic pressures were maintained.[34] Inducing IVS ischemia and dysfunction resulted in cardiac failure.[34] The administration of Dopamine improved left heart and IVS function, and subsequent right ventricular performance despite persistent experimental RV ischemia. Infusion of Dopamine resulted in hemodynamics that exceeded baseline.[34] Dopamine is an agent proven to improve hemodynamics in the presence of PHTN and RVSD.[18,33,34,38]

In an animal model, high doses of Dopamine (32 g/kg/min) increased MAP, mPAP, and cardiac index but did not significantly change SVR, PVR, or SAP/PAP.[40] Management of PHTN with RVSD requires multiple considerations. To enhance the benefits of Dopamine, one might consider the co-administration of a vasodilator.[29] In 27 patients with severe LV failure, Dopamine increased cardiac index from 1.8 to 2.5 L/min/m2, MAP from 83 to 89mmHg, mPAP from 37 to 44mmHg, but decreased PVR by 25%.[29] Combining Dopamine with Nitroglycerin increased cardiac index to 2.9 L/min/m2, while reducing pulmonary pressures, PVR, and central venous pressures.[29] In patients with pre-existing high left ventricular filling pressure with or with pulmonary hypertension, the combined effects of Dopamine with Nitroglycerin increase cardiac index and reduce pulmonary vascular resistance and PVR/SVR.[30]

When considering the best inotrope for managing PHTN and RVSD, inotropy, maintaining systemic perfusion pressures, and increasing SVR/PVR are important for successful management. Considering that RV contractility may not improve, management is directed toward the optimization of ventricular interdependence.[34,35] To improve RV-PA coupling, maintaining, or lowering PVR and raising SVR/PVR is important. Although reducing PVR

is ideal, pulmonary vascular reactivity, defined by a reduction in mPAP by 10 mmHg and/or achieving an mPAP less than 40 mmHg, is not guaranteed and may occur in as little as 10-20% of patients.[41-44] Reported responses to pulmonary vasodilators vary, including reductions in mPAP of only 9-10 +12 % and reductions in PVR by only 12-16 + 18-34%.[41,45] Given the variability of pulmonary vascular response, an inotrope that increases contractility while sustaining or increasing SVR and SVR/PVR will positively affect IVS position and improve systemic perfusion pressures and coronary perfusion. Considering this, Dopamine may be the best choice for managing PHTN with RVSD with or without the addition of Nitroglycerin or a different pulmonary vasodilator.[46]

Comparing Inotropes

It is difficult to draw conclusions regarding inotropes across studies as many variables exist, including methodology, dosing, and patient selection. Nevertheless, if it is assumed that all inotropes increase contractility and cardiac index equally, a focus on other hemodynamic data is simplified.[47] Across several receptor types, Dopamine is reported to cause mild vasodilation at very low doses, increase MAP, SVR, stroke volume at intermediate or higher doses, and heart rate at higher doses.[42] There is compelling evidence that Dobutamine, Epinephrine, and Norepinephrine have a wide range of adverse effects that can be undesirable in patients with RVSD and PHTN.

Dobutamine increases heart rate significantly and has a mixed effect on blood pressure, such that 10-20% of patients will experience hypotension and/or intolerable arrhythmias.[42,48,49] Dobutamine infusion of only 5 g/kg/min increased the vasoplegic state in 2/3 of patients with septic shock.[48] In a pulmonary artery banding model in dogs treated with dobutamine, increases in the cardiac index were related to increases in heart rate and not in stroke volume.[50] In a rabbit pulmonary hypertension model, dobutamine infusion (2-20 g/kg/min) decreased blood pressure, SVR, and SVR/PVR ratio, all of which are undesirable for IVS position and shape, and for coronary perfusion.[51] In a review of 9 studies involving pediatric patients, both Dopamine and Dobutamine increased cardiac index and systemic blood pressure with minimal or no changes in SVR, PVR, or cardiac filling pressures.[52]

Dopamine increased heart rate in two studies with infusion rates > 8 g/kg/min in one study and > 15 g/kg/min in a second. 76 Dobutamine increased heart rate in 2 of 4 studies between doses of 1-10 g/kg/min, while a third study recorded intolerable dysrhythmias and hypotension.[52] In patients with systemic sclerosis and pulmonary hypertension, Dobutamine infusion at 5 g/kg/min caused an increase in pulmonary artery pressure and a mild increase in cardiac index but had no significant impact on SVR/PVR.[53] The increase in cardiac index was again due to an increase in heart rate and not an increase in stroke volume.[53] In cardiac surgical patients with volume overload due to valvular regurgitation, a cross-over comparison of Dopamine and Dobutamine up to 5 g/kg/min reported comparable increases in heart rate, reduced PVR, and increased cardiac index.[54] While Dopamine increased blood pressure, Dobutamine reduced it, and both inotropes reduced the calculated SVR.[54]

Dopamine reduced PVR more and had a more favorable increase in SVR/PVR.[54] In an infant study involving systemic hypotension and poor peripheral perfusion, Dopamine (10-20 g/kg/min) had greater success in increasing systemic blood pressure, while Dobutamine (10-20 g/kg/min) significantly increased the heart rate.[55] After pulmonary artery banding resulted in increased RV pressure, reduced cardiac index, and systemic hypotension, Dobutamine (> 5 g/kg/min) increased heart rate and cardiac index, had little change on PVR but lowered SVR, leading to an increased PVR/SVR ratio.[36]

In infants/neonates, both Dopamine and Epinephrine increase blood pressure.[56] However, Epinephrine was associated with greater increases in heart rate, greater hyperglycemia, and lactic acidosis, requiring more bicarbonate and insulin.[56] Similar results were reported in a septic shock population comparing Dopamine to Epinephrine, the latter complicated by hyperglycemia and lactic acidosis.[57] Epinephrine may impair splanchnic circulation in

shock states,[58] while Dopamine increases hepatic and portal blood flow.[59] Although hemodynamic effects of Dopamine and Epinephrine appear similar, the metabolic effects of Epinephrine are undesirable.

Milrinone, an inodilator, may be the vasoactive agent of choice for many clinicians in the setting of PHTN and RVSD, but the evidence is not as compelling to support its use. In an isolated heart model with fixed loading conditions, milrinone failed to increase cardiac output compared to Dopamine.84 Maximal doses of Dopamine were shown to be superior to maximal doses of Milrinone in terms of increased contractility.[60] Studies have also shown that milrinone's vasodilating (both systemic and pulmonary) properties are its main effect, with little or no effect on inotropy and lusitropy.[61] In nine adults without cardiac disease scheduled for elective surgery, Milrinone resulted in an increased heart rate, decreased MAP, and decreased SVR.[62] The pulmonary capillary wedge pressure, cardiac index, and PVR did not change significantly.[62] When Dopamine was added, there was a reduction of the heart rate, an increase in MAP, and an increase in cardiac index.[62] In critically ill patients with pulmonary hypertension, Dopamine is a better first-line therapy to improve cardiac function and maintain systemic perfusion pressures.[25]

Criticism Of Dopamine

Most of Dopamine's backlash comes from studies claiming increased mortality associated with sepsis and increased pulmonary pressures and pulmonary vascular resistance.[63-65] De Backer et al compared Dopamine to Norepinephrine in a large, randomized study and found that Dopamine was associated with twice the incidence of tachyarrhythmias, mainly atrial fibrillation.[65] Otherwise, mortality was not different.[65] By contrast, a retrospective review of 520 patients with cardiogenic shock, Dopamine, and Norepinephrine reported similar occurrences of atrial (12.2 vs. 15.7% respectively) and ventricular (19.9 vs. 25.3% respectively) arrhythmias.[66] In a Medline search over 35 years, atrial tachyarrhythmias were similarly found in patients receiving Dopamine and Epinephrine.[67]

Milrinone and Dobutamine have been reported to be equally arrhythmogenic to each other and more pro-arrhythmic than Dopamine.[68] In patients with cardiogenic shock, both Milrinone and Dobutamine increased the heart rate by nearly 40%.[69] Arrhythmias were more common in patients treated with Dobutamine, which was the reason for discontinuation of the drug in 11.3% of cases.[69] While systemic hypotension occurred similarly in the two groups, it was a reason to discontinue Milrinone in 13.1% of cases.[69] Sato et al similarly reported that Dobutamine, Epinephrine, and Milrinone were nearly four times more likely to cause atrial fibrillation with rapid ventricular response compared to those not receiving inotropes.[70] More so, patients receiving Dobutamine, or Epinephrine were 2.5x and 5x more likely to die, respectively.[70] In another large database analysis, Dobutamine was associated with lower MAPs, higher heart rate, and significantly higher mortality.[71]

There is little denying that Dopamine improves hemodynamics in patients with septic and/or cardiogenic shock. In a controlled septic shock model, Dopamine increased cardiac index by 15%, systemic blood pressure by 20-30%, SVRi, and PASP by 10% but did not significantly increase mPAP.[70] Although Norepinephrine increased systemic blood pressures and resistance, it also increased PASP, mPAP, and PVR significantly more in association with a reduction in cardiac index.[46] In patients with septic or cardiogenic shock, Dopamine increased heart rate by 10-15%, MAP by 30%, stroke volume by 21-30%, and urine output.[30] Left ventricular diastolic pressures rose 2-4%, and central venous pressure declined.[30]

Mortality benefits vary equally for all inotropes.[70] In 417 septic patients, neither Dobutamine, Epinephrine, or Milrinone were associated with greater ICU-free days or lower mortality.[70] Despite improving ventricular contractility and hemodynamic data, the administration of inotropes is associated with increased mortality.[68,72,73] In 219 patients with cardiogenic shock, Dobutamine and Epinephrine were associated with significantly

greater mortality, the latter with greater renal insufficiency, and higher lactate levels.[74] In the same study, Dopamine was not associated with greater mortality.[74] In a database analysis (n=34,381) of patients with cardiogenic shock, inotropes were associated with up to 2x greater mortality. Mortality was higher with Dobutamine, Epinephrine, and Norepinephrine when doses exceeded 0.1 g/kg/min and with Dopamine > 15 g/kg/min.[75]

Conclusion

For patients with PHTN and RVSD, Dopamine provides a safe and superior inotrope option compared to dobutamine, milrinone, and epinephrine. Dopamine's effects on the pulmonary vasculature are dose-dependent, with vasodilation reported at lower infusion rates and minimal changes at higher infusion rates, all while reducing central venous pressure. By comparison, systemic pressures and resistance increase significantly, raising the SVR/PVR ratio. The minimal impact on pulmonary vascular resistance may reflect the absence of pulmonary vascular reactivity, while increases in mPAP reflect increases in pulmonary blood flow to an abnormal non-reactive pulmonary vascular tree. These improvements in forward flow are associated with a reduction in central venous pressure. Dopamine improves ventricular contractility and ventricular interdependence.

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