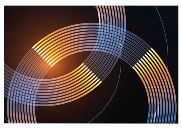
SCA NEWS

IN THIS ISSUE

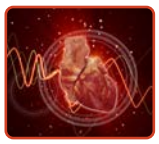
Volume 52 • Number 35 • February 2026

President's Message



SCA Echo

Thoracic Anesthesia Symposium



Annual Meeting and Workshops

Boots 'n Bling

SCA Endowment

Funding Opportunities

A&A Free Access

Member Elections

Member Spotlight

Our History

Residents & Fellows Corner

Echo Corner Free CME

Literature Reviews

Pro / Con Debate

SCA
Society of Cardiovascular Anesthesiologists

PRESIDENT'S MESSAGE

SOCIETY OF CARDIOVASCULAR ANETHESIOLOGISTS

Dear Colleagues,

As we head into a new year, it is a good time to think about all the exciting possibilities for the SCA in 2026 and beyond. I will devote several upcoming President's Messages to describing the great work being done by the SCA's committees regarding excellent research and educational offerings.

Amanda Fox MD, MPH

President
Society of Cardiovascular Anesthesiologists

In this message, however, I want to update you about some growth and opportunities in global activities of the SCA. The SCA's Mission Statement is "Transforming perioperative cardiovascular and thoracic care through education, research and global collaboration." In the spirit of such collaboration, I have invited Dr. Alex Mittnacht, MD, and inaugural President of the newly founded International Academy of Cardiac Anesthesiologists (IACA) to co-author this Message with me. Dr. Mittnacht was previously the Chair of the International Council of the SCA, and this partnership offers opportunities to advance the SCA"s global approaches to increasing knowledge and patient care. As such, please consider all that follows as a jointly written statement between us in our Presidential roles!

We owe a debt of gratitude to the SCA's founding members, who envisioned SCA as an international organization. It is because of the SCA's exemplary leadership over the years that the SCA is widely recognized as a leader in cardiac anesthesia. From the SCA's early days, its mission was carried out with this broader scope in mind. The designation as an international organization was, in large part, exemplified by the representation of international partner societies at the committee and board of directors level, as well as by organizing the biennial International Congress of Cardiac Anesthesiologists (ICCVA).

The idea of a global cardiac anesthesia community with a shared vision of collaboration and leadership in education, research, quality, and safety improvement in cardiac anesthesia ultimately led to the creation of the International Academy of Cardiac Anesthesiologists (IACA). The SCA is one of the founding societies, and it is important to note that IACA does not in any way compete with national societies' interests and autonomy but rather serves as a shared hub for international outreach and collaboration. Membership is through the national societies and special interest groups, rather than individual membership.

IACA serves to enhance its member societies' visibility on the global stage and promotes shared vision and mission goals. The concept of

PRESIDENT'S MESSAGE

an international biennial meeting will continue, now organized with equal input from all partner societies. We just had the inaugural meeting in Sydney, Australia, hosted by the Australian & New Zealand College of Anesthetists (ANZCA). The meeting was a resounding success, with attendance and participation by a global cohort of cardiac anesthesia representatives, including many SCA members. The next meeting will be in Rome, Italy, in September 2027, organized together with the European Association of Cardiothoracic Anesthesia and Intensive Care (EACTAIC) as the host society.

The SCA now has representation on the IACA board of directors and members on every IACA committee. Going forward, projects emerging from the various committees will offer significant opportunities for SCA members to get involved.

Another way for you to get involved with SCA's global initiatives is through the International Council (IC). The SCA IC is currently chaired by Dr. Stan Shernan (Chair) and Dr. Pablo Motta (VC) and reports directly to the SCA Executive Committee. Members of the IC comprise SCA members-at-large and nominees from partner societies. The SCA IC serves as the main point of contact for our international partner societies, and its leadership is represented at the IACA.

Many SCA members have enjoyed the hospitality extended to us by our partner societies since the SCA's founding. We have now established an International Symposium for our international colleagues to share their expertise at the SCA annual meeting. During this symposium, colleagues from around the world will present on topics of interest to the global cardiac anesthesia community.

There is a lot to learn from each other, and there is no doubt in our minds that we are stronger, and can achieve more, together.

With gratitude,

Amanda Fox, MD
SCA President, 2025-2027

SCA ECHO 2026

February 26 - March 1, 2026
Atlanta, Georgia

SOCIETY OF CARDIOVASCULAR ANESTHESIOLOGISTS
Knowledge • Care • Investigation

Alina Nicoara MD, FASE Co-Chair

Charles Nyman MBBCh, FASE Co-Chair

Kimberly Howard-Quijano MD, MS, FASE Vice-Chair

Message from the Program Chairs

We are thrilled to invite you to SCA Echo 2026, taking place February 26 – March 1, in the vibrant city of Atlanta, Georgia!

Get ready for four immersive days of learning, collaboration, and inspiration as we explore the cutting edge of echocardiography in perioperative and surgical care. The 2026 program will expand on the series of dynamic multidisciplinary panels that explore the pivotal role of echocardiography in shaping surgical decision-making, especially in the realms of valvular heart disease, mechanical circulatory support and data discordance.

Step into the heart of the operating room as we tackle real-world clinical challenges and evolving strategies. Participate in enriching discussions on the latest transcatheter procedures in structural heart disease, and the implications of the recent guidelines.

Elevate your expertise in our signature "Learn from the Experts" sessions which will highlight echo-anatomic correlations with porcine dissections of the aortic, mitral and tricuspid valves.

Whether you're aiming to refine your skills, expand your clinical toolbox or connect with leaders in the field, **SCA Echo 2026** offers an unmatched opportunity to achieve your goals.

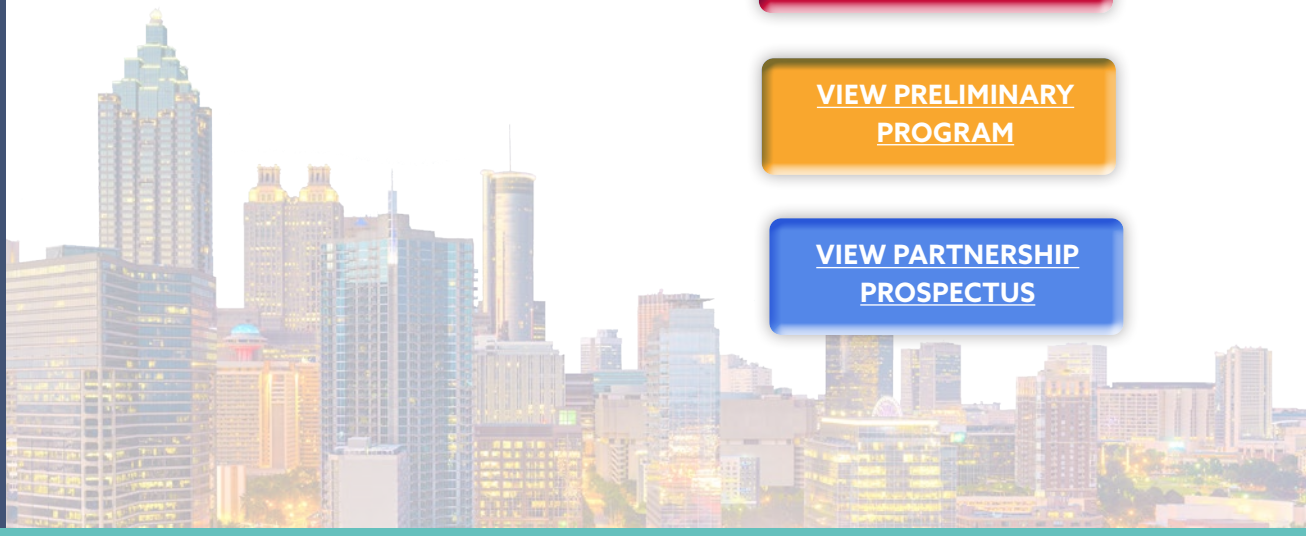
In-person and Virtual Registration Is Now Open!

Alina Nicoara, MD, FASE – Co-Chair
Charles Nyman, MBBCh, FASE – Co-Chair
Kimberly Howard-Quijano, MD – Vice-Chair

REGISTER NOW

VIEW PRELIMINARY PROGRAM

VIEW PARTNERSHIP PROSPECTUS



Registration is Now Open!

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:

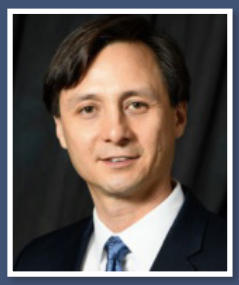
- Choose 3 in-person workshops and register for an optional live PBLD for a conference experience tailored to YOUR educational needs.
- Network with 200 other professionals in anesthesiology to help you gain insight into your practice and career.
- Connect with our exhibitors to learn about new products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

REGISTER NOW | **VIEW PRELIMINARY PROGRAM** | **VIEW PARTNERSHIP PROSPECTUS**

Meeting is Now on Thursday!

SCA ANNUAL MEETING & WORKSHOPS

April 23-26, 2026 • Nashville, Tennessee



Jonathan Ho
MD, FASE
Chair

Stephanie Ibekwe
MD, MBA, MPH, MS
Vice-Chair

SCA2026

Registration is Now Open!

Look forward to:

- Amazing content delivered by experts in cardiothoracic anesthesiology, interventional cardiology, and cardiothoracic surgery.
- Experts will provide didactics, small group breakout teaching, and high yield discussions.
- Problem based learning discussions, scientific abstracts, and workshops are planned to optimize attendee learning and connection on critical cardiothoracic anesthesiology topics.
- Attendee networking, idea-sharing, and exhibits

This year, in-person you can:

- Attend live discussion sessions to help you discover up to date practice pathways and innovations in the field
- Register for Workshops and PBLDs tailored for YOUR educational needs
- Network with 1,200 other professionals in anesthesiology as well as thoracic surgeons to help you gain insight into your practice and career
- Connect with industry and exhibiting companies to learn about new products and programs

REGISTER NOW | **VIEW PRELIMINARY PROGRAM** | **VIEW PARTNERSHIP PROSPECTUS**

New Meeting Pattern – Thursday through Sunday!

Registration is Now Open!

BOOTS AND BLING BASH

Friday, April 24, 2026 ▪ Country Music Hall of Fame

Celebrate With Us!

This will be an unforgettable evening filled with great company, live entertainment, dancing, and plenty of Nashville flair. The **Boots & Bling Bash** will be a special event and a wonderful opportunity to celebrate the SCA community, reconnect with colleagues and friends, and support the mission that unites us.

Whether you're a returning member or joining the celebration for the first time, this event will deliver a night of laughter, music, and memories you won't want to miss.

Purchase Your Tickets Today!

Event Details

Date:
Friday, April 24, 2026

Time:
7:00 PM

Location:
Country Music Hall of Fame

Live Music By Tanglewood

Energetic Country Rock Band from Nashville

CLICK HERE to Purchase Your Ticket to This Amazing Event!

Grab your cowboy hat, dust off your boots, and shine up your bling for the SCA Boots & Bling Bash in Nashville, Tennessee!

SCA ENDOWMENT

Your Support is Important

Dear SCA Member,

The SCA exists to advance education, leadership, and research—and this work is made possible by individuals who care deeply about our mission. We would like to take a moment to highlight our accomplishments from the past year and share what lies ahead in 2026.

2025 IN REVIEW

2025 marked significant progress for the SCA Endowment, reflecting our members' strong commitment to the Society's future:

- We established a **planned giving program**, enabling members to include the SCA in their estate planning.
- We successfully launched the **Marianne and Michael Cahalan Legacy Circle**, ensuring perpetual funding for an annual mid-career research grant.
- We strengthened the **Kaplan Leadership Award**, expanding it to two awardees and celebrating recipients with a dedicated reception at the Annual Meeting.
- We introduced the **Jane Fitch Leadership Award**, with its inaugural cycle beginning in 2026.
- We now update the **Donor Honor Roll** regularly and offer a concierge experience at the Annual Meeting for donors at upper giving levels.
- We began publishing an annual **Endowment Report**, providing transparency into how gifts are invested and stewarded with fiscal responsibility.
- We further strengthened our long-standing partnerships with **Veritas**, our management company, and Modera, our investment firm of more than 20 years — helping ensure thoughtful growth of every donation.

LOOKING AHEAD TO 2026

This year promises even more impact. With your continued support, we will fully launch the Jane Fitch Leadership Award, engage a carefully selected and diverse group of leaders in award selection, and expand support for fellows both nationally and internationally. As a member of this vibrant and cohesive Society, there will always be meaningful opportunities to give, participate, and celebrate our shared mission.

Thank you again for all that you do to support the SCA. We hope you will consider making a donation to help power the Society forward.

With warm regards,

The SCA Endowment Team
The SCA — Powered by You

DONATE TODAY

GRANTS

2026 SCA Research Grants — *Apply Now!*

SCA supports cardiothoracic and vascular research projects. This is the basis for the creation of the **SCA Starter Grant, Joyce Wahr Starter Grant, Michael Cahalan Grant, and the In-Training Grant.**

Grant Information

Four types of grants will be awarded in 2026:

- SCA Michael Cahalan Grant – up to $75,000 per year for two years.
- SCA Starter Grant – up to $37,500 per year for two years.
- SCA Joyce Wahr Starter Grant – up to $37,500 per year for two years.
- SCA In-Training Grant - $15,000 for one year.

Please Note: The Starter Grant and the Joyce Wahr Starter Grant request the same application information and formatting. At the time of application, the PI should identify if they are eligible for, and wish to be considered for, both the Joyce Wahr Starter Grant and the Starter Grant, or for one or the other only.

The awards will be announced prior to the 2026 SCA Annual Meeting & Workshops in Nashville, TN. The grant period of 24 months can begin any time from July 1 to December 31 of the year granted. Grant recipients are required to present their work at a subsequent SCA Annual Meeting.

CLICK HERE

Applications will Close on February 9, 2026 —

START YOUR RESEARCH APPLICATION NOW!

Click Here for the 2026 SCA Research Grants requirements and instructions.

Click Here for the Research Grants Checklist (Please submit with your application).

Click Here for the Research Grants Title Page (Please submit with your application).

ANESTHESIA & ANALGESIA®

How to View Free Access Articles

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

Cardiovascular and Thoracic Anesthesiology

Cardiovascular Pathophysiology and Outcomes

Hemostasis and Thrombosis

SCA 2026 Elections – Voting is Now Open!

The 2026 online elections for SCA leadership positions are open through March 10. The candidates are running for the following positions: Active and Associate members in good standing will receive a personalized link to their primary and secondary email address to cast their vote.

- **DIRECTOR-AT-LARGE**
- **EARLY CAREER DIRECTOR**

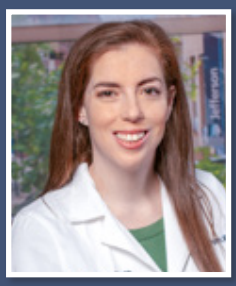
The SCA Nominating Committee, chaired by Immediate Past President Dr. Kathy Glas, is pleased to endorse the following candidates for the 2026 election cycle:

Good Luck to Our Nominees

DIRECTOR-AT-LARGE

Andra Duncan, MD
Cleveland Clinic

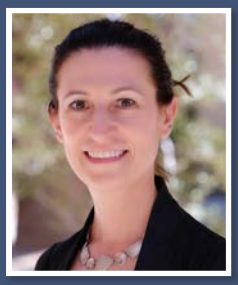
Dr. Duncan is currently serving as an SCA Director-at-Large and is seeking reelection. Dr. Duncan has practiced cardiothoracic anesthesia at the Cleveland Clinic for the past 24 years. In addition to her role on the SCA Board, she has served on the SCA Program and Research committees and as a Board liaison for the Member Engagement and QSV committees and SCA/STS database subcommittee. Dr. Duncan has also served on ASA, ASE, and AATS cardiac anesthesia committees. She is an Associate Editor for the SCA-affiliated journal, Anesthesia & Analgesia. Dr. Duncan is active in clinical research and has received funding from NIH, SCA, and industry. She enjoys mentoring junior investigators in research designed to improve and prolong the lives of patients undergoing cardiac surgery. Her goal as a Director-at-Large is to listen and support the members of the cardiac anesthesia community and advance the mission of the SCA.



Gina Linganna, MD
University of Pennsylvania

Dr. Linganna is an associate professor at the Hospital of the University of Pennsylvania and is pursuing her master's in medical education. 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 and the ARC Question Bank. She currently serves on the Board of Directors as an Early Career Member. Additionally, she currently chairs the Online Education Committee and is responsible for the content of SCA University. She has also broadened the scope of the Society's collaboration with outside organizations such as the Society of Thoracic Surgeons. Dr. Linganna hopes to be elected to the Board of Directors to continue her SCA contributions on this large scale.

DIRECTOR-AT-LARGE



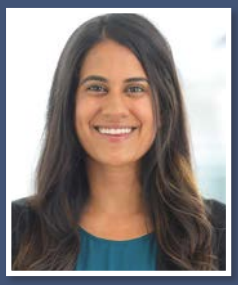
Alessia Pedoto, MD, FASA
Memorial Sloan-Kettering Cancer Center

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

Jochen Steppan, MD, DESA, FAHA, FASA
Johns Hopkins University School of Medicine

Jochen Steppan is an Associate Professor at Johns Hopkins University, doing adult and pediatric cardiac anesthesia. He serves as the Director for Perioperative Medicine, High Risk Cardiovascular Disease. He completed medical school at Heidelberg in Germany, and he did his residency and fellowships at Johns Hopkins. Dr. Steppan served the SCA for almost 20 years. He is a founding member and former Chair of the 'Cardiovascular Outcomes Research in Perioperative Medicine (COR-PM) Conference', now part of the SCA meeting to advance clinical cardiovascular outcomes research, focusing on mentorship of junior members, and using an inclusive and diverse approach. He served on the Research Committee, International Committee, and the Scientific Program Committee. Dr. Steppan has published over 90 peer reviewed publications and delivered over 60 national and international talks. His research on the molecular mechanisms underlying pulmonary hypertension, HFpEF, and aging has been funded by FAER, SCA, AHA, and the NIH.

EARLY CAREER DIRECTOR



Serena S. Dasani, MD, MBA
UT Health Houston

Dr. Dasani is a cardiothoracic anesthesiologist, assistant professor, and health services researcher at UT Health Houston. Her trajectory began as a Fulbright scholar and followed with an MD/MBA at the University of Pennsylvania. She then completed her residency and fellowship at Brigham and Women's Hospital where she served as Chief Resident. At UT Health Houston, her work integrates management science, echocardiography, and value-base care research. Her scholarship appears in prominent journals often through cross-institutional collaborations with SCA colleagues. She has been active in the SCA since training, earning the inaugural Women in cardiothoracic Anesthesia (WICTA) Fellow of the Year award, SCA In-Training Grant, and SCA Early Career Investigator award. She serves on the SCA's Economic & Governmental Affairs Sub-Committee and WICTA's Executive Committee as the Research and Writing Liaison. Above all else, she combines societal engagement, leadership acumen, and investment in mentorship to advance the field of cardiac anesthesia globally.

Hesham Ezz, MD
Dartmouth Geisel School of Medicine

Dr. Ezz an adult cardiothoracic and critical care anesthesiologist currently staffing the Cardiothoracic operating rooms and Cardiovascular ICU at Dartmouth Health Medical Center as an Assistant Professor. After graduating from Tanta University Medical School, Egypt in 2015, he joined Yale University, Applied Hemodynamics department as a post-doctoral associate. From there Dr. Ezz joined Anesthesiology residency at Yale New Haven Hospital where he served as a chief resident 2021-2022. He then chased his subspecialty interests in cardiothoracic and vascular anesthesiology at the Cleveland Clinic, OH and critical care at the University of Pennsylvania Hospital, PA. Dr. Ezz is very passionate about education and leadership and have a special clinical interest in the perioperative care of heart failure and aortic surgical patients and the utilization of mechanical circulatory support in shock and respiratory failure patients.

Sergey Karamnov, MD
University of Massachusetts Medical Center

Dr. Karamnov, is a cardiovascular and thoracic anesthesiologist who serves as Division Chief of Thoracic Anesthesia and Associate Director of Research at the University of Massachusetts Memorial Medical Center, and Associate Professor of Anesthesiology at the University of Massachusetts Chan Medical School. He is deeply committed to training and mentorship, having guided more than 60 residents, fellows, and medical students over the past nine years, many of whose work has been honored with competitive awards and presented on national stages. During his tenure at Brigham and Women's Hospital, he cofounded and directed the Adult Cardiothoracic Anesthesiology Fellow Research Program (ACARP), which has produced over a dozen SCA award recipients, has been highlighted numerous times in JCVA, and has gained national recognition as a model for structured research training. A recipient of the SCA Starter Grant and SCA Kaplan Leadership

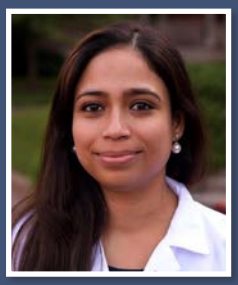
Development Award, Dr. Karamnov continues to build platforms that strengthen mentorship, foster collaboration, and expand opportunities for early-career anesthesiologists.

EARLY CAREER DIRECTOR

Aibek Mirrakhimov
University of Kentucky

Dr. Aibek Mirrakhimov is an Assistant Professor of Anesthesiology, Perioperative, Critical Care, and Pain Medicine at the University of Kentucky, where he also serves as Director of Integrative Transesophageal Echocardiography (TEE) and Point-of-Care Ultrasound (POCUS), PACU Rotation Director, and Assistant Residency Program Director for POCUS. He is board-certified in Internal Medicine, Critical Care Medicine, General Anesthesiology, and Adult Cardiac Anesthesiology, and holds National Board of Echocardiography certifications in Advanced Perioperative TEE and Critical Care Echocardiography.

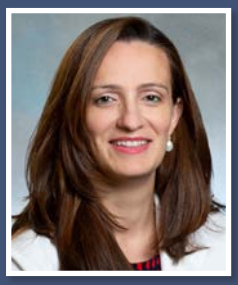
Dr. Mirrakhimov has completed extensive postgraduate training across internal medicine, critical care, anesthesiology, and adult cardiothoracic anesthesiology at institutions including the University of New Mexico and Cleveland Clinic. Widely regarded as a skilled educator and mentor, he has organized regional POCUS conferences and developed numerous academic lectures and digital educational resources. He is an active member of the Society of Cardiovascular Anesthesiologists and the American Society of Anesthesiologists.



Samhati Mondal, MD
University of Maryland School of Medicine

Dr. Mondal is an Associate Professor at the University of Maryland School of Medicine. She serves as the Division Chief of Transplant Anesthesiology and Co-Director of Medical Student Education in the department of Anesthesiology. Dr. Mondal completed medical school from the University of Calcutta. She has completed two anesthesiology residencies – from the University of Delhi followed by from MetroHealth, Case Western Reserve University. Additionally, she pursued two advanced fellowships in Liver Transplant and Cardiothoracic anesthesiology from the Cleveland Clinic and The Johns Hopkins University, respectively. Her research focuses on outcomes in cardiothoracic and transplant anesthesia, particularly in Enhanced Recovery After Cardiac Surgery and regional anesthesia applications and perioperative coagulation management well-aligned with her clinical fields of cardiac and transplant cases. She is committed to translating this work into evidence-based practices that improve patient care. Dr. Mondal has authored more than 45 manuscripts, spearheaded much outcomes research as principal and co-investigator and led industry sponsored clinical trials at her site.

EARLY CAREER DIRECTOR



Gabriela Querejeta Roca, MD
Brigham & Women's Hospital

Dr. Querejeta Roca is an Assistant Professor of Anesthesiology at Harvard Medical School and an attending physician at Brigham and Women's Hospital (BWH). She serves as Medical Director of Perioperative Transesophageal Echocardiography (TEE) and Associate

Program Director of the Adult Cardiothoracic Anesthesiology Fellowship, roles that allow her to combine clinical innovation, program development, and mentorship. After training in Spain, Dr. Querejeta Roca joined BWH, where she completed a research fellowship and a cardiothoracic anesthesia fellowship. Her research on sleep apnea, pulmonary hypertension, and cardiac imaging has been published in leading journals. At BWH, she led the effort to achieve one of the first national accreditations in perioperative TEE by the IAC. She continues to advance imaging innovations while training the next generation of cardiothoracic anesthesiologists. Beyond her institution, she contributes to the field through service on national committees, including the Society of Cardiovascular Anesthesiologists' Echo Week planning group.

Patrick Upchurch, MD
University of Utah

Dr. Upchurch built his entire career around understanding cardiopulmonary physiology, caring for patients in the cardiac OR and cath lab, ensuring immediate safety and optimizing durable success by providing exceptional care and imaging in the context of sound clinical judgement. After completing his residency and three fellowships in critical care, cardiothoracic anesthesiology, and perioperative echocardiography, he joined the faculty in the Department of Anesthesiology, Perioperative and Pain Medicine at the University of Utah School of Medicine. Locally, he currently serves as the Director of Structural Heart Imaging and Associate Program Director of the Adult Cardiothoracic Anesthesiology fellowship. Nationally, he is a member of the ASA Committee on Patient Blood Management, the ASA Educational Track Subcommittee on Cardiac Anesthesia, and the SCA Blood Management Subcommittee. Dr. Upchurch academic interests include structural heart imaging guidance, patient blood management, and enhanced recovery after cardiac surgery.

SPOTLIGHT

Awesome Woman Interview

Diana Anca, MD

Associate Professor of Clinical Anesthesiology
Weill Cornell
New York

Dr. Diana Anca is an Associate Professor of Clinical Anesthesiology at Weill Cornell in New York. She completed her Cardiothoracic Anesthesia fellowship at Columbia Presbyterian Medical Center. Dr. Anca is currently the Director of NORA at Weill Cornell. Her clinical and academic interests include Electrophysiology and Interventional Pulmonology. She is the Workshop and PBLD Coordinator and the incoming Vice-Chair for Thoracic Anesthesia Symposium.

What led you to become a Cardiovascular/Thoracic Anesthesiologist?
I was drawn to cardiothoracic anesthesia early on during my residency, by the combination of physiology, complexity of tasks and skills needed to navigate caring for such patients, the need for procedures (invasive monitoring, TEE), and the teamwork involved. Being part of a truly multidisciplinary team from the pre-procedure until ICU and discharge created a blueprint for me for all other areas of my journey. It is fascinating to see the ever-evolving field of cardiothoracic anesthesia, aligned with the advancement of structural heart procedures, Interventional Pulmonology, and Electrophysiology, and our versatility and evolution of anesthesiologists.

How did you hear about the SCA?
I had heard about the SCA during my residency, but it wasn't until my fellowship that I attended my first meeting together with my co-fellows and attendings, and I think I only missed couple of meetings during my career. Through collaborations with my cardiothoracic anesthesia colleagues from across the country and internationally, it led to a great community of anesthesiologists engaged in the advancement of cardiothoracic anesthesia.

What roles have you held for the society?
I currently serve as the Workshop and PBLD Coordinator for the Thoracic Anesthesia Symposium (TAS), incoming Vice-Chair for the next couple of years starting in April 2026, and I am part of the Sub-Committee of Atrial Fibrillation. As part of TAS Leadership, I participate in organizing the annual meeting and Workshops, as well as the TAS panel for SCA annual meeting and the TAS at EACTAIC. I am looking forward to a great meeting in Nashville in April 2026.

What is one of your greatest achievements as a Cardiovascular/Thoracic Anesthesiologist?
During my career as a cardiothoracic anesthesiologist I participated actively in presentations at the SCA and TAS annual meetings, both as a speaker and organizer of TAS, but I am particularly proud of my engagement and collaboration with EACTAIC, the SCA European counterpart, as well as my participation as invited speaker at AHA and HRS, where I was able to outline the role of anesthesiologists in the team. I am also actively involved in SCA University.

Do you have any advice for fellows and residents?
This is a field where preparation and teamwork are very essential, so invest early on the fundamentals, seek mentors who will both challenge you and advocate for you, and commit to lifelong learning. Interact and learn from the other team members, surgeons, proceduralists, nurses, perfusionists, and keep in mind the less traditional "cardiothoracic" procedures, as structural heart procedures, interventional pulmonology,

radiology and Electrophysiology claim an ever-growing part of the cardiothoracic field.

On a practical note, SCA has a lot of resources for fellows: fellow sessions, abstracts, a mentoring circle, SCA University, WICTA, so make use of them, and in the process develop connections with peers and outside mentors.

Have you experienced any difficulties as a woman in the field?
In a high-complexity and acuity field such as cardiothoracic anesthesia there were of course challenges, and it took work to be recognized, but I think this is where strong mentors and teamwork and building trust within the team plays an important role. Also, the Women in CTA (WICTA) within the SCA is an important mentorship, leadership building resource, and I can see that during my career there are more women in leadership roles in cardiothoracic anesthesia, and I can honestly say that it's not a "male -dominated" field anymore.

Do you have any advice for other women in the field?
I think it is a layered approach: master your craft and trust your training, select strong and inspiring mentors, but also stay connected with like-minded peers, engage within your team at institutional level and the SCA/TAS, WICTA, which are great resources. Don't forget to pay it forward, mentor fellows and junior colleagues.

How do you balance work and personal life?
While both work and personal life might be different for different people, it's important to be honest with yourself, practical, set up boundaries, and keep in mind that goals, interests and priorities might change during your career, so be flexible.

What is something you enjoy doing outside of work?
I love opera and theater, which I have great opportunities to enjoy living in New York City, I try to see as many Broadway shows as I can, and I am a frequent presence at the Metropolitan Opera, I love travelling but stick with urban travelling and not the more wild, adventurous one. And of course, shopping!

Would you change anything about the path you took to get to where you are now?
While I wouldn't change my path, I would perhaps be more selective on some on the journey's steps, especially during training and early career, enjoy activities that at that time I thought I don't have the time or ability to do. The one silver lining in my path is forging great collaboration with the teams I worked in the institutions I practiced and developing lifelong friendships and mentorships.

What was the best piece of advice you received?
Be versatile, develop a niche within the specialty and don't turn down opportunities because you might feel uncomfortable with new tasks or responsibilities.

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Did you know...

- The SCA was incorporated in Louisiana in 1978, and its first Annual Meeting took place in November 1979.
  Check out the SCA's History Timeline to learn more!
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Surgical Aortic Valve Replacement and Acute Coronary Ostial Occlusion: A Case Study

Authors: Adam Kohutnicki, MD MBA MS; Zach Woodward DO; Greg Morrisette, MD

Introduction

Bicuspid aortic valve is the most common congenital aortic valve anomaly, with an incidence purported in the literature of 1-2% with a male predominance. Congenital bicuspid aortic valves are associated with various valvular pathologies, including aortic stenosis and aortic insufficiency. Typically patients with bicuspid aortic valves are asymptomatic until 50-60 years of age depending on severity of associated valvular pathology. Severe aortic stenosis, defined by the American College of Cardiology and American Heart Association (ACC/AHA) as an orifice area less than 1.0 cm, a mean gradient greater than 40 mmHg, or jet velocity greater than 4 m/s. Severe aortic stenosis is fatal with a mortality rate of 50% over 2 years with a poor prognosis unless the valve is replaced.[1] There are several known risks to surgical aortic valve replacement. These include acute ostial stenosis, plaque rupture, clot or plaque embolization, vasospasm, and need for re-replacement.[2] More complex surgeries, such as combined aortic valve and aortic root replacement with an aortic valved conduit, introduces similar risks, namely with placement of the coronary button. In this case study, we introduce a planned combined coronary artery bypass graft and aortic valve replacement in which the aforementioned risks were realized, including coronary ostial occlusion, severe left and right ventricular dysfunction, and ultimately advanced mechanical circulatory support.

Case Presentation

A 51-year-old female with a past medical history of coronary artery disease with non-obstructive first obtuse marginal stenosis, hypertension, hyperlipidemia, obesity and known nonrheumatic bicuspid aortic valve presented to the clinic with worsening dyspnea, chest tightness, and dizziness. Subsequent workup found severe aortic stenosis with a mean gradient of 44 mmHg and valve area of 0.9 cm2. Preoperative left heart catheterization was notable for 50% stenosis of the first marginal branch. The patient was taken to the operating room for coronary artery bypass grafting to the first obtuse marginal artery and aortic valve replacement. After installation of a 23mm bioprosthetic valve and OM1 CABG, attempted separation from CBP was complicated by significant left ventricular dysfunction and EKG abnormalities suggestive of ischemia. The heart was re-arrested and the bioprothetic valve was found to be effacing the left coronary ostia. A 23mm aortic valved conduit was installed secondary to poor aortic root quality that was deemed unable to attempt a second bioprosthetic valve implantation, thus aortic root replacement was required in addition to coronary button implantation. Upon the second attempted separation from CPB and after IABP placement, severe right ventricular dysfunction was found, thought to be secondary to a right coronary button issue, and CPB was resumed. Rather than redoing the button, a saphenous vein graft was used to bypass the RCA at the level of the right atrial appendage. Upon completion of surgery, transesophageal echocardiography revealed normal left ventricular function and mild right ventricular dysfunction. The patient was brought to the cardiothoracic intensive care unit on inotropic and vasopressor support, intra-aortic balloon pump with 1:1 support, atrio-ventricular pacing with underlying sinus rhythm, intubated and sedated. The patient was extubated later that day, weaned from mechanical circulatory support post operative day (POD) one , weaned from vasopressors and transferred to the floor on POD two. On POD five the patient experienced angina symptoms and coronary angiography revealed a proximal bend with resulting stenosis of the SVG-RCA graft for which two drug eluding stents were placed. Later that day she had a recurrence of similar symptoms however angiography was unremarkable. She was discharged two days later on POD seven.

Discussion

There are few case reports discussing acute coronary stenosis as a complication of surgical aortic valve replacement. Previous case reports often identify thromboembolic causes as the

culprit for acute coronary occlusion secondary to atrial fibrillation, atrial myxomas, ventricular thrombus, or calcific embolism from calcific aortic stenosis.[3]

In this case, following the initial attempted wean from bypass, the etiology of severe left ventricular dysfunction was thought to be a result of left main coronary ostia occlusion from the initial bioprosthetic valve. The heart was re-arrested and the aorta was reopened which showed the new bioprosthetic aortic valve effacing the left main coronary ostia, the likely culprit for acute left ventricular dysfunction seen coming off of bypass. The surgeon found at this time the aortic root to be of poor quality and it was decided to replace the aortic root with an aortic valve and root conduit.

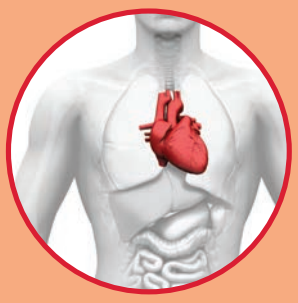
Following the second attempted wean from bypass, it was observed that the heart despite inotropic support was struggling, and the decision was made to place an intraaortic balloon pump via a previously placed femoral arterial line under transesophageal echocardiogram guidance. Despite aggressive de-airing of the heart, it became apparent the right ventricle was struggling. The etiology of severe right ventricular dysfunction was not initially clear. During transesophageal echocardiography, it was observed that there was significant air in the left ventricle, so it was believed that significant air had entered the right coronary artery. Initial surgical evaluation with doppler flow was supportive of a patent right coronary artery; however, due to the right ventricle continuing to show evidence of dysfunction on transesophageal echocardiography as well as in the surgical field, it was surmised that there must be an issue with the right coronary artery. It was later suggested the doppler was inappropriately picking up right ventricular flow as opposed to right coronary artery flow. Rather than re-arrest the heart, an off-pump right coronary artery bypass was attempted, however, this was abandoned due to perceived difficulty of finding a suitable target. The heart was re-arrested and cardiopulmonary bypass was initiated for a third time.

Upon opening of the right coronary artery, it was observed that there was no significant antegrade flow, confirming suspicion for right coronary ostia occlusion from the aortic valve conduit coronary button. A single graft was created to the right coronary artery, establishing antegrade flow, and observed immediate improvement in right ventricular function. With inotropic and mechanical circulatory support, the patient was successfully weaned from bypass and was brought to the cardiovascular intensive care unit.

This case highlights not only severe possible complications associated with aortic valve replacement but also the role of transesophageal echocardiography, closed loop communication between the anesthesia and surgical teams, and utilization of mechanical circulatory support.

Works Cited

1. Rajput FA, Zeltser R. Aortic Valve Replacement. 2023 May 29. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan–. PMID: 30725821.
2. Jang M-S, Choi J-H, Han J-H, Choi YI, Kim J-M, Youn HC. Acute coronary artery obstruction after aortic valve replacement surgery and role of transesophageal echocardiography. Anesth Pain Med. 2017; 12(4): 348-351. https://doi.org/10.17085/apm.2017.12.4.348
3. Fernández, A. L., El-Diasty, M. M., Martínez, A., Alvarez, J., & García-Bengochea, J. B. (2011). A simple technique to rule out occlusion of right coronary artery after aortic valve surgery. The Annals of thoracic surgery, 92(6), 2281-2282.



ECHO CORNER

LEARNER NOTIFICATION

Society of Cardiovascular Anesthesiologists
Activity Title: 2026 SCA Echo Corner (Beyond Vegetations, Diagnosing Aortic Valve Masses)
Release Date: 2/2/2026
Expiration Date: 2/2/2028
Activity Type: Enduring Material

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

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

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

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

Educational Information

Physician Practice Gap:

Despite routine use of echocardiography, many clinicians may lack sufficient knowledge to accurately differentiate papillary fibroelastoma from other benign and malignant cardiac masses, including Lambl excrescences and primary malignant cardiac tumors, based on tumor prevalence, typical valve involvement, and anatomic location. This gap may lead to diagnostic confusion, unnecessary concern for malignancy, or failure to recognize papillary fibroelastoma as the most common valvular tumor of the aortic valve and the most common left-sided cardiac tumor.

Cardiothoracic anesthesiologists may have limited understanding of the distinct transesophageal echocardiographic characteristics of papillary fibroelastomas, including their pedunculated morphology, narrow stalk, homogeneous speckled appearance, and differentiation from thin, filiform Lambl excrescences. Limited familiarity with these imaging features may impair accurate diagnosis and intraoperative decision-making, particularly when evaluating small, mobile valvular masses.

Needs that Underlie the Gap

There is a need to provide education to clinicians on epidemiology, anatomic predilection, and differential diagnosis of cardiac valvular masses, particularly to recognize papillary fibroelastoma as the most common benign valvular tumor of the aortic valve and to distinguish it from rarer malignant cardiac tumors and non-neoplastic entities such as Lambl excrescences.

There is a need to further educate clinicians on the echocardiographic identification of papillary fibroelastomas, including their characteristic pedunculated morphology with a narrow stalk appearance on TEE, and differentiation from thin, filiform Lambl excrescences, to improve diagnostic accuracy and guide appropriate clinical and surgical decision-making.

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

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

- evaluate common cardiac neoplasms and their etiology
- identify echocardiographic characteristics specific to different types of cardiac masses
- discuss clinical presentations of cardiac masses, including neurologic events

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

Contact Information
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ECHO CASE: Beyond Vegetations, Diagnosing Aortic Valve Masses

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CASE PRESENTATION

A 78-year-old female with hypertension, type 2 diabetes, thyroid nodules, ductal carcinoma in situ of the right breast s/p resection, BMI of 35.0kg/m2, moderate aortic stenosis (AS) with long standing dyspnea on exertion and 11mm x 9mm cardiac mass on the left coronary cusp who presents for robotic excision of aortic valve mass and aortic valve repair. Pre cardiopulmonary bypass transesophageal echocardiogram is shown below.

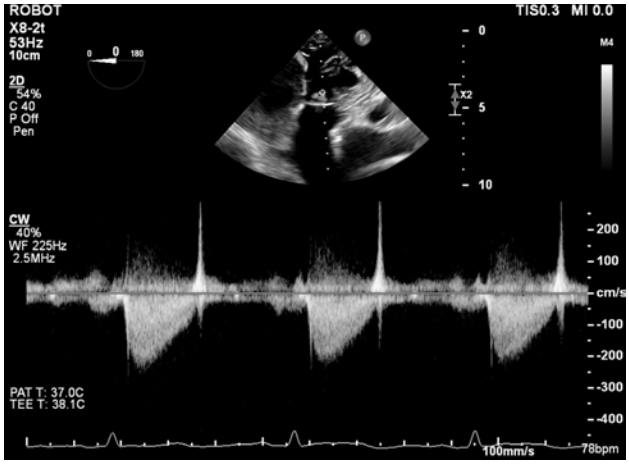
WATCH VIDEO 1 WATCH VIDEO 2 WATCH VIDEO 3

Question 1: What is the most likely differential diagnosis for this patient's aortic valve mass?

A) Angiosarcoma
B) Papillary fibroelastoma
C) Mesothelioma
D) Lambl Excrescences

Moderate to severe aortic stenosis is noted on TEE. A mobile echodensity measuring 11mm x 5mm was seen attached to the left coronary cusp. The appearance is most consistent with that of a papillary fibroelastoma.

WATCH VIDEO 4



Question 2:
What are key characteristics of a papillary fibroelastoma on TEE?

A) Thin filiform strands
B) Highly vascularized mass
C) Pedunculated lesion with a narrow stalk
D) All of the above

3D imaging of the valve and papillary fibroelastoma is obtained. See image on next page.

WATCH VIDEO 5

WATCH VIDEO 6

Question 3: **Where on the valve are fibroelastomas most likely found?**

A) Downstream side of cardiac valves on valvular commissure
B) Upstream side of cardiac valves on valvular commissure
C) Upstream side of cardiac valves on valvular endocardium
D) Downstream side of cardiac valves on valvular endocardium

Question 4: **What symptoms can patients with papillary fibroadenomas present with?**

A) Stroke
B) Myocardial infarction
C) Ventricular fibrillation
D) All of the above

This patient underwent robotic excision of aortic valve PFE and valve sparing aortic valve cusp repair with no anesthetic or surgical complications.

Question 5: **What post-excision findings should be considered?**

A) Recurrence of new lesion
B) New embolic event
C) Valvular dysfunction
D) All of the above

Figure 1. Lambl excrescence and PFE

ANSWERS

Question 1:

ANSWER: (B) Papillary fibroelastoma

75% of cardiac tumors are benign tumors including cardiac myxomas, lipoma, papillary fibroelastoma and fibromas. Of the benign tumors, papillary fibroelastoma (PFE) is most found on aortic valves and the most common left sided tumor (Figure 1). Papillary fibroelastoma is the most common valve tumor involving the aortic valve (44%).

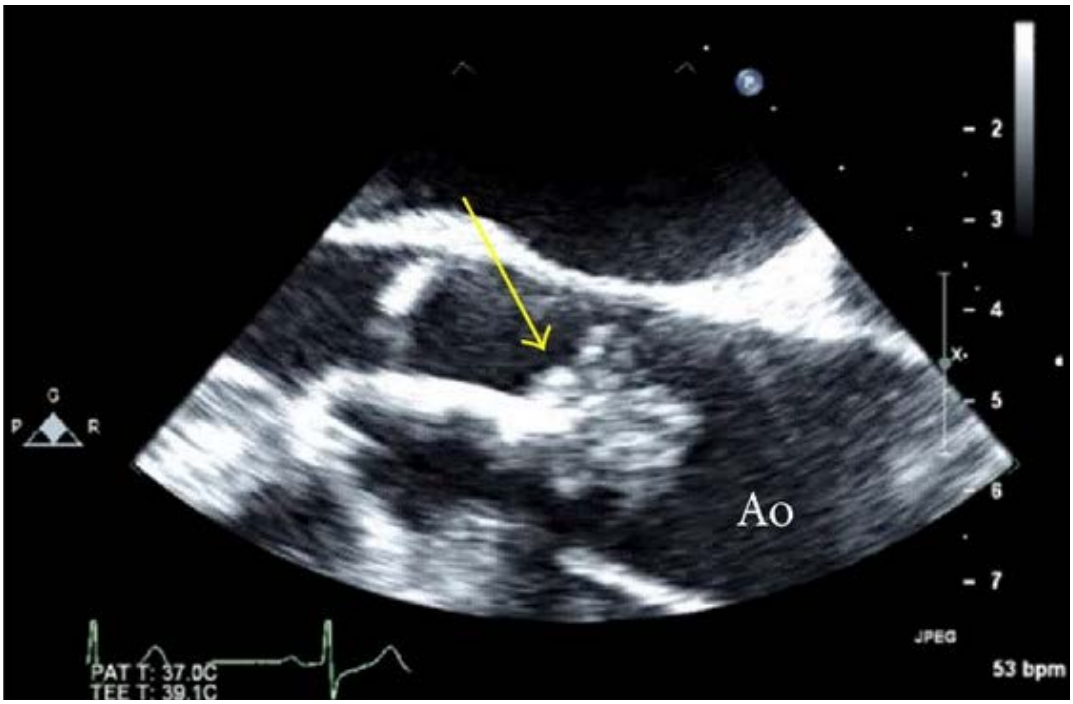
Primary malignant cardiac tumors are rare. These include angiosarcoma, rhabdomyosarcoma, mesothelioma and fibrosarcoma. These are primarily found in the right atrium or pericardium and rarely affect the aortic valve. Angiosarcoma usually involve blood vessels like the pulmonary artery or aorta. Lambl excrescences are degenerative fibrous strands originating from areas of valve coaptation (Figure 1). It should not be confused with tumors or vegetations. Aortic and mitral valves are the two most common locations of Lambl excrescences.

Question 2:

ANSWER: (C) Pedunculated lesion with a narrow stalk

On TEE, PFEs have a typical "pom-pom" homogenous speckled appearance on echo and is seen as a pedunculated lesion with a narrow stalk. This is different to lambl excrescences which are seen as thin, hypermobile and filiform strands (Figure 2).

The gross appearance of PFE is classically described as a "sea anemone" with a central stalk and frond-like arms projecting outwards. These tumors can range from 2mm to several cms in dimension. Histologically, PFEs are composed of collagen, elastin and reticulin with minimal vasculature. The outer layer is comprised of the endothelium, an intermediate later with mucopolysaccharide-rich connective tissue, and a central core of fibrin and mucopolysaccharide. Lambl excrescences are smaller and non-branching. They also have less abundant subendothelial myxoid ground substance.



Question 3:

ANSWER: (D) Downstream side of cardiac valves on valvular endocardium

Papillary fibroelastoma most commonly arises on the endocardial surfaces of heart valves, with a predilection for the aortic and mitral valves, but can also affect the tricuspid and pulmonary valves. Fibroelastomas typically originate on the downstream aspect of a valve (e.g., the aortic side of the aortic valve).

Question 4:

ANSWER: (D) All of the above

There are several postulated etiologies for PFEs. PFEs are thought to be lesions that begin as microthrombi that join at sites of endothelial damage. These PFEs have the potential to grow as microthrombi join which can potentially embolize leading to multiple complications. Stroke, myocardial infarction, ventricular fibrillation and sudden death are amongst these complications. Cerebral arteries are the most affected by embolization which can lead to transient ischemic attacks and strokes.

Sudden cardiac death from myocardial infarction and ventricular fibrillation can occur due to prolapse of a tumor into the coronary ostium or coronary occlusion by emboli from the aortic valve.

Question 5:

Answer: (D) All of the above

Recurrence of papillary fibroelastoma after surgical excision has been documented and highlights the need for ongoing postoperative surveillance with echocardiography, particularly transesophageal echocardiography. Recurrence rates may be higher in patients who initially presented with embolic events, and recurrent lesions can occasionally be mistaken for Lambl excrescences, necessitating careful imaging and, if indicated, histopathologic confirmation.

Valve-sparing excision is usually preferred, with low operative mortality and excellent long-term outcomes, but patients should be monitored for potential valvular dysfunction or regrowth at the excision site. The risk of embolic events is significantly reduced after complete resection, but patients with a history of preoperative embolic events may have a higher risk of recurrence and warrant closer follow-up.

References

1. 1. Devanabanda AR, Lee LS. Papillary Fibroelastoma. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan.
2. Kondamareddy D, Masood W. Lambl Excrescences. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan.
3. Daveron, E., Jain, N., Kelley, G.P., Luer, W.H., Fermin, C., Helmcke, F. and Kerut, E.K. (2005), Papillary Fibroelastoma and Lambl's Excrescences: Echocardiographic Diagnosis and Differential Diagnosis. Echocardiography, 22: 461-463.
4. Carino D, Nicolini F, Molardi A, Indira Dadamo C, Gherli T. Unusual Locations for Cardiac Papillary Fibroelastomas. J Heart Valve Dis. 2017 Mar;26(2):226-230.
5. Khair T, Mazidi P, Laos LF. Cardiac Papillary Fibroelastoma: case report and review of the literature. Int J Cardiol. 2010 Feb 18;139(1):102-4.
6. Sorour AA, Kurmann RD, El-Am EA, Bois MC, Scott CG, Lee AT, Dearani JA, Maleszewski JJ, Klarich KW. Recurrence of Pathologically Proven Papillary Fibroelastoma. Ann Thorac Surg. 2022 Apr;113(4):1208-1214.
7. Mazur P, Kurmann R, Klarich KW, Dearani JA, Arghami A, Daly RC, Greason K, Schaff HV, Ahmad A, El-Am E, Sorour A, Bois MC, Viehman J, King KS, Maleszewski JJ, Crestanello JA. Operative management of cardiac papillary fibroelastomas. J Thorac Cardiovasc Surg. 2024 Mar;167(3):1088-1097.e2.
8. Fine, Nowell M., Foley, David A., Breen, Jerome F., Maleszewski, Joseph J., Multimodality Imaging of a Giant Aortic Valve Papillary Fibroelastoma, Case Reports in Medicine, 2013, 705101, 3 pages, 2013.

Impact of Propofol or Sevoflurane on the Renoprotective Effect of Remote Ischaemic Preconditioning in Cardiac Surgery: The HypnoRenalRIP Randomized Clinical Trial

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Zarbock, A., Schöne, L.M., Kellum, J.A., Gerss, J., Weiss, R., Böke, H., & Meersch, M. (2025).

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Background

Acute kidney injury (AKI) is one of the most frequent and severe complications following cardiac surgery, affecting up to 30–80% of patients depending on the definition used.[1] AKI is linked to increased mortality, prolonged hospital stays, chronic kidney disease, and long-term dialysis dependency. Given its major impact on outcomes and healthcare costs, strategies to prevent AKI are a high clinical priority.[2,3]

Remote ischemic preconditioning (RIPC)—brief cycles of limb ischemia and reperfusion before surgery—has been proposed as a non-invasive, low-cost organ protection strategy.[4] Earlier studies, including the RenalRIP trial, showed that RIPC could reduce AKI incidence in high-risk patients by inducing transient increases in renal stress biomarkers, reflecting cell cycle arrest and activation of protective pathways.[5,6]

However, other large multicenter RIPC trials failed to reproduce these benefits, possibly because most patients received propofol-based anesthesia, which experimental data suggest might suppress the preconditioning response by blocking mitochondrial or inflammatory signaling.[7,8] The HypnoRenalRIP trial by Zarbock et al. (2025) was therefore designed to test whether propofol attenuates, and sevoflurane preserves, the renoprotective effects of RIPC during cardiac surgery in high-risk patients.

Summary

This single-center, double-blind randomized clinical trial investigated whether the type of anesthetic—propofol or sevoflurane—influences the renoprotective effects of remote RIPC in high-risk cardiac surgery patients. The study enrolled 160 patients undergoing cardiopulmonary bypass (CPB), randomized into four groups: propofol+RIPC, propofol+sham-RIPC, sevoflurane+RIPC, and sevoflurane+sham-RIPC.[9]

The primary outcome was the change in urinary biomarkers, indicators of renal stress and cell cycle arrest—before and after RIPC. Results showed that RIPC triggered a significant increase in these biomarkers only under sevoflurane anesthesia (median increase 0.070 vs. −0.015 with propofol, P = 0.022).[9]

Although no statistically significant differences were observed in clinical outcomes such as AKI, dialysis dependency, or mortality, patients anaesthetized with sevoflurane exhibited trends toward improved renal biomarker profiles and fewer AKI cases (30% vs. 45% in propofol+RIPC). The findings indicate that propofol may suppress the molecular signaling necessary for RIPC-induced renoprotection, likely by inhibiting the release of high mobility group box protein-1 (HMGB-1), a key mediator of preconditioning pathways.[9]

Strengths

1. **Robust randomized design:** The study's 2×2 factorial, double-blind structure minimizes bias and allows independent assessment of both anesthetic and RIPC effects.
2. **Mechanistic insight:** Measurement of cell cycle arrest biomarkers (9·9) provides a validated and mechanistically relevant surrogate for early renal stress.
3. **Novel clinical contribution:** This is the first human RCT demonstrating that propofol can inhibit the renoprotective biochemical response to RIPC.

4. **Blinded analysis and control:** The trial maintained blinding at all levels—care providers, data collectors, and analysts—enhancing the credibility of results.9

Limitations

1. **Single-center design:** Conducted at one German hospital, limiting external generalizability to other populations and surgical settings.
2. **Limited statistical power:** The trial's 75% power (below the conventional 80%) and modest sample size may have obscured significant differences in patient-centered outcomes like AKI rates.
3. **Biomarker surrogate endpoint:** Primary outcomes were biochemical rather than clinical, meaning renal protection was inferred, not directly observed.
4. **Uncontrolled confounders:** The use of thiopental in sevoflurane groups might have introduced an uncontrolled pharmacologic influence on biomarker expression.
5. **Short-term follow-up:** Although 90-day outcomes were reported, longer-term renal function effects remain unassessed.[9]

Conclusions

RIPC triggered transient renal stress biomarker responses only under sevoflurane, not propofol, suggesting that propofol suppresses the cellular mechanisms responsible for RIPC-induced renal protection. While no significant clinical differences in AKI incidence were observed, the biomarker data provide compelling evidence that anesthetic choice influences the success of organ-protective preconditioning strategies.[9]

Clinical Relevance to Practice

- **Anesthetic selection matters:** When RIPC is intended as a renoprotective strategy during cardiac surgery, propofol-based anesthesia should be avoided, and volatile agents like sevoflurane are preferred.
- **RIPC feasibility:** Given its low cost and simplicity, RIPC remains a practical intervention, but its effectiveness is highly anesthetic-dependent.
- **Biomarker-guided monitoring:** Measurement of urinary 9·9 could be integrated into perioperative protocols to identify patients likely to benefit from RIPC or early renal support strategies.
- **Future implications:** Larger multicenter studies focusing on hard clinical outcomes are warranted to validate whether sevoflurane-based RIPC translates into reduced postoperative AKI and mortality.

References

1. Hobson CE, Yavas S, Segal MS, et al. Acute kidney injury is associated with increased long-term mortality after cardiothoracic surgery. Circulation. May 12 2009;119(18):2444-53. doi:10.1161/CIRCULATIONAHA.108.800011
2. Priyanka P, Zarbock A, Izawa J, Gleason TG, Renfurm RW, Kellum JA. The impact of acute kidney injury by serum creatinine or urine output criteria on major adverse kidney events in cardiac surgery patients. J Thorac Cardiovasc Surg. Jul 2021;162(1):143-151.e7. doi:10.1016/j.jtcvs.2019.11.137
3. Lafrance JP, Miller DR. Acute kidney injury associates with increased long-term mortality. J Am Soc Nephrol. Feb 2010;21(2):345-52. doi:10.1681/ASN.2009060636
4. Zarbock A, Schmidt C, Van Aken H, et al. Effect of remote ischemic preconditioning on kidney injury among high-risk patients undergoing cardiac surgery: a randomized clinical trial. JAMA. Jun 02 2015;313(21):2133-41. doi:10.1001/jama.2015.4189
5. Rossaint J, Meersch M, Thomas K, et al. Remote ischemic preconditioning causes transient cell cycle arrest and renal protection by a NF-κB-dependent Sema5B pathway. JCI Insight. Jul 22 2022;7(14)doi:10.1172/jci.insight.158523

6. Hausenloy DJ, Candilio L, Evans R, et al. Remote Ischemic Preconditioning and Outcomes of Cardiac Surgery. N Engl J Med. Oct 08 2015;373(15):1408-17. doi:10.1056/NEJMoa1413534
7. Meybohm P, Bein B, Brosteanu O, et al. A Multicenter Trial of Remote Ischemic Preconditioning for Heart Surgery. N Engl J Med. Oct 08 2015;373(15):1397-407. doi:10.1056/NEJMoa1413579
8. Kottenberg E, Thielmann M, Bergmann L, et al. Protection by remote ischemic preconditioning during coronary artery bypass graft surgery with isoflurane but not propofol - a clinical trial. Acta Anaesthesiol Scand. Jan 2012;56(1):30-8. doi:10.1111/j.1399-6576.2011.02585.x
9. Zarbock A, Schöne LM, Kellum JA, et al. Impact of propofol or sevoflurane on the renoprotective effect of remote ischaemic preconditioning in cardiac surgery: the HypnoRenalRIP randomised clinical trial. Br J Anaesth. Dec 2025;135(6):1626-1634. doi:10.1016/j. bja.2025.08.055

Perioperative Tight Glucose Control Regimens for Preventing Surgical Site Infections following Cardiac Surgery: A Systemic Review and Metanalysis of Randomized Controlled Trials

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Sun et al, revisited the issue of tight (TGC) versus conventional glucose management during cardiac surgery using surgical site infections (SSI) as the primary end point. Secondary end points analyzed include hypoglycemia, length of ICU stay, neurological deficits and all-cause mortality within 30 days after surgery.

Using the Preferred Reporting Items for Systemic Reviews and Metanalyses checklist they were able to find 26 RCTs fitting the inclusion criteria. Any type of cardiac surgery in patients older than 18 was included and only studies after 1990. Other criteria included studies with TGC target levels less than or equal to 150 mg/dL, studies with subgroup data available or if the cohort has at least 50% of participants to be eligible.

The study concluded that SSI risk is significantly reduced during cardiac surgery if TGC is initiated at the start of surgery. In terms of secondary outcomes, there is an increased risk of hypoglycemia regardless of patient diabetic status and the timing of the initiation of tight glucose control. If TCG was initiated before surgery, ICU stay was reduced for patients with diabetes but patients without diabetes showed no change. TCG did not demonstrate any correlation for postoperative neurological deficits or all-cause mortality.

Strengths

- Strong design of a metanalysis study
- Narrow inclusion criteria for definitive objectives
- Clearly defined outcomes both primary and secondary

Limitations

- Multiple studies across different institutions included had varied methodologies resulting in different features such as definitions of TGC or patient factors, follow-up timing and control protocols.
- Many studies did not use SSI as primary outcome leading to low powered study and results
- Perioperative factors that could have relevance were unrecorded such as nutrition/ nutritional status, antibiotic use and conditioning status.

Clinical Relevance to Practice

Sun et all confirms what prior studies have, TCG is not appropriate for all patients in all scenarios. Even with the close monitoring during the operative period, there are increased risks of severe hypoglycemia. TCG does provide benefits when started before surgery and this is generally possible as the anesthesiologist will be monitoring the patient very closely during the operative period. Further investigation would need to be conducted to see if these benefits would continue if the TGC was not continued in the post-operative period.

Additional studies should also be conducted once continuous glucose monitoring is more available. Theoretically, this could make tighter glucose control safe and potentially decrease the risk of severe hypoglycemia in specific patient groups continues to be high.

References

Perioperative Glycemic Management in Cardiac Surgery: A Narrative Review Thongsuk, Yada et al. Journal of Cardiothoracic and Vascular Anesthesia, Volume 38, Issue 1, 248 – 267

Tight versus liberal blood-glucose control in the intensive care unit: special considerations for patients with diabetes von Loeffelholz, Christian et al. The Lancet Diabetes & Endocrinology, Volume 12, Issue 4, 277 - 284

Strict versus liberal target range for perioperative glucose in patients undergoing coronary artery bypass grafting: A prospective randomized controlled trial Desai, Shalin P. et al. The Journal of Thoracic and Cardiovascular Surgery, Volume 143, Issue 2, 318 - 325

Perioperative Care in Cardiac Surgery: A Joint Consensus Statement by the Enhanced Recovery After Surgery (ERAS) Cardiac Society, ERAS International Society, and The Society of Thoracic Surgeons (STS) Grant, Michael C. et al. The Annals of Thoracic Surgery, Volume 117, Issue 4, 669 - 689

Restrictive Versus Liberal Oxygenation in Patients Undergoing Cardiopulmonary Bypass-assisted Heart Surgery: A Randomized Controlled Trial

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Summary

This is a single-center, patient and assessor-blinded randomized trial of adult patients undergoing coronary artery bypass grafting (CABG) , aortic valve replacement (AVR), or both, utilizing cardiopulmonary bypass (CPB.) Patients were randomly assigned in a 1:1 fashion to a "restrictive" oxygenation group in which the Fio2 during and for the first hour after weaning from CPB was maintained at 50%, compared to the "liberal" group in which the Fio2 was maintained at 100%, the standard of care. The primary composite outcome was the time to death, stroke, renal failure requiring dialysis, or new-onset or worsening heart failure. 1389 patients were randomized with a mean age of 67 years and only 17% female, with a median follow-up period of 5.9 years (IQR, 2.5 – 8.3.) 24% of each group met what the authors call "the primary endpoint" with a hazard ratio of 1.01 (95% confidence interval 0.8 – 1.3, P=0.92) The authors conclude, unsurprisingly, that "Among patients undergoing elective or urgent CPB-assisted coronary artery bypass grafting, aortic valve replacement, or both, no significant differences were observed in mortality, dialysis-dependent renal failure, stroke, or new-onset or worsening heart failure between a restrictive oxygenation strategy (Fio2 50%) and a liberal oxygenation strategy (Fio2 100%) during CPB and the subsequent weaning period."

Strengths

- Randomized Methodology
- Large patient population
- Single-center study ensuring homogeneity of methods and care.

Limitations

- In the Methods section the authors mention that this trial was part of a larger study, the "Glorious Trial" in which patients were assigned randomly to either receive, or not receive, a GLP-1 analogue exenatide, in addition to being randomized to the liberal versus restrictive oxygenation protocol.
- The authors assert that "The two interventions were a priori and assumed to be independent from each other..." (emphasis in the original.) They state that there was no interaction between the GLP-1 intervention and the oxygen trial with a Pinteraction = 0.40, but this data is not presented.
- The studies listed as references for damaging effects of hyperoxia are not convincingly clinically relevant.
- The data is highly skewed towards males. Even as of 2016 The Danish Heart Surgery registry was reporting that 27% of people undergoing heart surgery in Denmark, the locale of the study, were women, significantly higher than the 17% reported in the current study.

- The authors' final paragraph summarizes the conclusion that most cardiac anesthesiologists already know "In summary, no differences were observed in mortality, dialysis-dependent renal failure, stroke, or new-onset or worsening heart failure between a restrictive oxygenation strategy (FIO2 50%) and a liberal oxygenation strategy (Fio2 100%) during CPB for CABG, AVR, or both."

Conclusions and Clinical Implications

Interestingly, 10% of the patients in the restrictive oxygenation group received an Fio2 of greater than 50% at some point during the intervention, most commonly due to an Spo2 below 92%. This highlights the fact that the period of weaning from CPB and immediately thereafter is the most challenging and fraught time interval of the surgery, and therefore adding another potential complicating factor, with no added benefit to the patient, is unwarranted. This study therefore confirms the safety and prudence of utilizing an Fio2 of 100% in our patients undergoing cardiac surgery utilizing CPB.

References

1. Özcan C, Juel K, Flensted Lassen J, von Kappelgaard LM, Mortensen PE, Gislason G. The Danish Heart Registry. Clin Epidemiol. 2016 Oct 25;8:503-508. doi: 10.2147/CLEP.S99475. PMID: 27822091; PMCID: PMC5094640.

Right Ventricle–Pulmonary Artery Coupling and Outcomes After Cardiac Surgery

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RRight ventricular (RV) dysfunction is an increasingly recognized determinant of adverse outcomes following cardiac surgery. Historically underemphasized relative to left ventricular performance, RV function has been shown to independently predict perioperative morbidity, prolonged recovery, and mortality across a range of cardiac surgical populations. Traditional echocardiographic indices of RV function, however, incompletely capture the complex interaction between RV contractility and pulmonary vascular load, prompting interest in RV–pulmonary artery (RV–PA) coupling as a more physiologically integrated metric.

RV–PA coupling describes the ability of the right ventricle to adapt its contractile performance to changes in pulmonary arterial afterload. Invasive assessment using pressure–volume loop–derived end-systolic elastance to arterial elastance (Ees/Ea) remains the gold standard but is impractical for routine clinical use. Consequently, noninvasive surrogates have been developed, most notably the ratio of tricuspid annular plane systolic excursion to pulmonary artery systolic pressure (TAPSE/PASP). This ratio integrates a measure of longitudinal RV systolic function with pulmonary vascular load and has been validated against invasive measurements in patients with pulmonary hypertension and heart failure.

A growing body of literature has established the prognostic value of TAPSE/PASP in non-surgical and transcatheter populations. Seminal work by Guazzi and colleagues first demonstrated that TAPSE/PASP reflects RV contractile reserve and predicts outcomes in heart failure. Subsequent studies confirmed its association with mortality, rehospitalization, and functional capacity across populations with pulmonary arterial hypertension, left-sided heart failure, and valvular disease. More recently, TAPSE/PASP has emerged as a robust predictor of outcomes following transcatheter aortic valve replacement, transcatheter mitral and tricuspid interventions, and venoarterial extracorporeal membrane oxygenation (VA-ECMO), with lower ratios consistently identifying patients with impaired RV–PA coupling and worse prognosis.

Despite this expanding evidence base, data on RV–PA coupling in patients undergoing open cardiac surgery have remained limited. Prior investigations have largely focused on isolated RV dysfunction or specific surgical subgroups, such as rheumatic multivalve disease, with limited generalizability. The study by Sun et al. meaningfully advances the field by evaluating the association between preoperative TAPSE/PASP ratio and postoperative outcomes in a large, contemporary cohort of patients undergoing coronary artery bypass grafting and/or valve surgery.

In this single-center retrospective analysis, a preoperative TAPSE/PASP ratio below 0.52 mm/mm Hg was independently associated with an increased risk of major morbidity and operative mortality, even after adjustment for Society of Thoracic Surgeons (STS) predicted risk and cardiopulmonary bypass duration. Importantly, the TAPSE/PASP ratio provided prognostic information incremental to established risk models, which do not incorporate echocardiographic measures of RV performance. Lower TAPSE/PASP ratios were also associated with clinically meaningful secondary outcomes, including prolonged intensive care unit stay, extended mechanical ventilation, and increased hospital length of stay, reinforcing the clinical relevance of RV–PA coupling beyond mortality alone.

This study aligns with prior transcatheter and heart failure literature demonstrating that RV–PA uncoupling reflects limited RV reserve and vulnerability to perioperative stressors. The use of invasively measured PASP from right heart catheterization strengthens the physiologic validity of the findings, particularly given known limitations of Doppler-derived RV systolic pressure estimates in patients with pulmonary hypertension or minimal tricuspid regurgitation. However, the high prevalence of pulmonary hypertension and valve surgery within the cohort suggests

that the identified cutoff may not be universally generalizable and should be interpreted within the clinical context.

Collectively, the findings by Sun et al. support the growing recognition of RV–PA coupling as a clinically meaningful, noninvasive marker of cardiopulmonary reserve. This work extends prior evidence by demonstrating its prognostic value in surgical cardiac populations and highlights the potential role of TAPSE/PASP in preoperative risk stratification. Future studies are needed to determine whether targeted optimization of RV function or pulmonary vascular load can modify risk in patients identified as having impaired RV–PA coupling before cardiac surgery.

PRO CON

INTRODUCTION OF PROS AND CONS:
Moderate vs Deep Hypothermia for Circulatory Arrest — TIME!!

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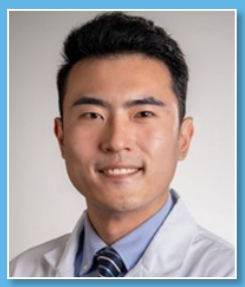

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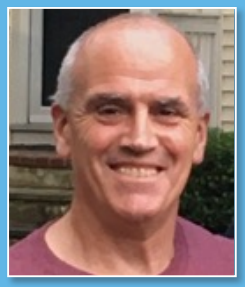

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Introduction

Circulatory arrest is a technique employed when cessation of blood flow is needed to achieve a bloodless surgical field and perform complicated surgical procedures. It is most often utilized during operations involving the aortic arch. Other surgeries that may utilize circulatory arrest include repairs of the descending thoracic aorta, complex congenital cardiac lesions, and pulmonary thromboendarterectomies. Infrequently, cardiac surgeries with a severely calcified, or 'porcelain aorta', may utilize circulatory arrest to avoid aortic cross clamping to reduce risk causing irreparable aortic injury and/or stroke.[1] Circulatory arrest is rarely considered for tumor resections involving the cava and/or right atrium or for complicated neurosurgical procedures involving large cranial aneurysms or in which massive air embolism is a risk.[2]

The cessation of oxygen and nutrient delivery, during circulatory arrest, to end organs leads to a cascade of events resulting from hypoxia and systemic inflammation, which, upon reestablishment of blood flow causes a reperfusion injury. While all organ systems are at risk for injury during circulatory arrest, central neurologic tissues are at extremely high risk even during brief periods of deprivation due to its high metabolic rate and lack of energy stores. Oxygen delivery and utilization are closely matched.

The brain represents 2% of the total body weight but utilizes 20% of the total body oxygen. It receives 15-20% of the total cardiac output.[3] Tissue hypoxia depletes adenosine triphosphate (ATP), causing a shift to anaerobic metabolism, resulting in a lactic acidosis and reduced cellular pH.[4,5] Neuronal hypoxic injury stimulates release of glutamate to open N-methyl-D-aspartate (NMDA) channels, increasing calcium influx, activating proteases and leading to mitochondrial dysfunction and apoptosis.[5] If continued, this leads to an excitotoxic cascade causing inflammation, free radical production, cell death, inflammation, disruption of the blood brain barrier, capillary leak and cellular edema.6 (Figure 1)

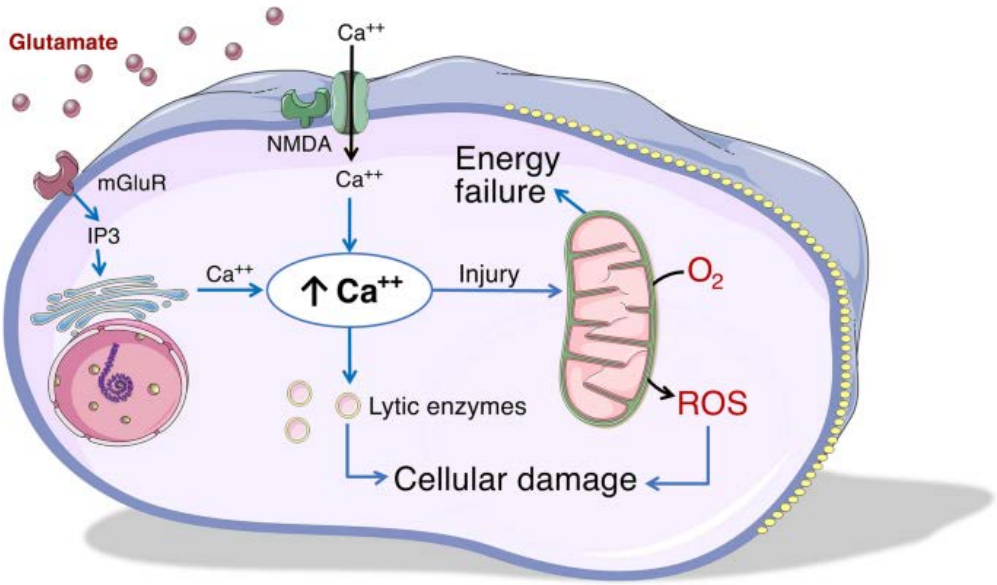

Figure 1 . Reproduced from Sandroni et al., 2021[6]

Clinically, neurocognitive injury can manifest as confusion/delirium, seizure, stroke, and death. The incidence of acute neurologic injury including stroke after aortic surgery with circulatory arrest ranges from 5-13%, with permanent complications reported in as many as 10-11%, and new pathology detected on MRI in up to 40%.[7-11] Postoperative cognitive dysfunction and stroke are directly related with duration of arrest.

Hypothermia

Defined as a temperature < 35°C, hypothermia can be accidental or therapeutic, the latter having been introduced into clinical medicine the 1950s to reduce oxygen (VO2) consumption and carbon dioxide production (VCO2). Hypothermia confers neuroprotection by lowering cerebral metabolic rate of oxygen (CMRO2). A 1°C decrease is associated with a decrease in cellular metabolism of 5-7%.[4,12] (Figure 2) Hypothermia decreases cellular metabolism and ATP breakdown, shifting the balance toward supply.[4] By improving oxygen supply/demand balance, hypothermia decreases the release of proinflammatory mediators, the development of reactive-oxygen species, ultimately protecting the blood-brain barrier from breakdown by metalloproteases reduces neuronal excitotoxic ischemic injury.[4]

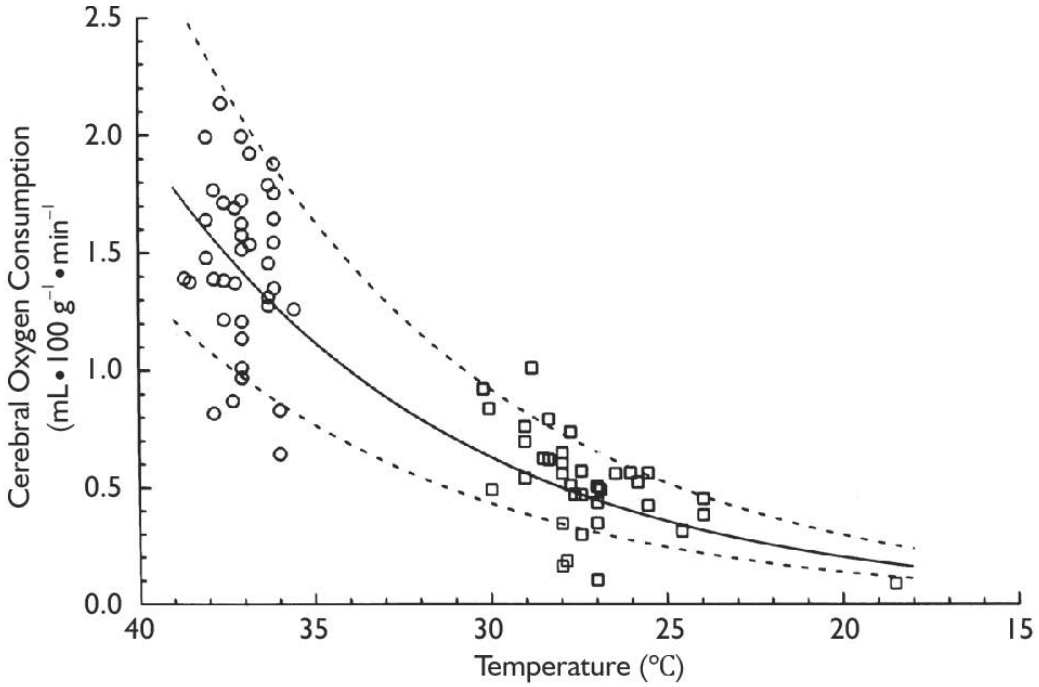

Figure 2. Kirklin JW, Barratt-Boyes BG. Cardiac surgery: morphology, diagnostic criteria, natural history, techniques, results, and indications. 2nd ed. New York: Churchill Livingstone; 1993.[12]

Even before use of CPB, surgical repairs of atrial and ventricular septal defects, and aortic and mitral valve replacements were performed during circulatory arrest after institution of systemic hypothermia (28-29°C).[13] In the 1970s, hypothermia was used for the first time for cerebral protection for aortic arch repair.[14]

The institution of hypothermia prolongs the duration of a 'safe' circulatory arrest to allow performance of complicated surgical procedures while preventing or minimizing end organ injury, with greatest focus on the brain and spinal cord. By reducing tissue metabolism, oxygen and nutrient requirements, hypothermia can attenuate end-organ injury during circulatory arrest. Previously published 'safe duration of hypothermia circulatory arrest' was derived from animal and human data, where CMRO2 was measured at varying body temperature. Although non-linear, and with reported variability, for every 10°C reduction (Q10) the metabolic rate declines by approximately 2.0-2.2x, halving oxygen consumption, for the brain and whole body (95% confidence interval 2-2.5x), or by approximately 50%.[15,16] When combined with cardiopulmonary

bypass and cardioplegia, hypothermia has permitted performance of complex cardiac surgical procedures that required longer cardiopulmonary bypass time, and/or those procedures requiring circulatory arrest. Most often hypothermia and circulatory arrest are utilized in combination during major aortic surgeries.

Experimentally, profound (10°C) core hypothermia allows circulatory arrest for 90 minutes in a canine model without developing motor or sensory disturbances.17 If the head was additionally immersed in an ice bath, circulatory arrest may be extended to two hours.[18]

For humans undergoing aortic surgeries, circulatory arrest times less than 20 minutes are considered safe when coupled with at least moderate hypothermia. Circulatory arrest times > 25 minutes are correlated with cognitive and fine motor impairment and longer hospital stays.[6] Based on the assumption that normothermic (37°C) circulatory arrest is tolerated for 5 minutes and measurements of cerebral metabolic rate and blood flow McCollough et al concluded CMRO2 and safe arrest times are different degrees of hypothermia (TABLE 1).15 In 37 adults scheduled for cardiac surgery during hypothermic circulatory arrest (HCA) authors concluded that a 'safe' time of HCA at 15°C is 29 minutes, and 40 minutes at 10°C.[15]

Table 1. Calculated safe intervals for interruption of brain perfusion at various temperatures*[15]

Temperature (°C)	Cerebral metabolic rate (% of baseline)	Calculated Safe duration (min)
37	100	5
30	56 (52-60)	9 (8-10)
25	37 (33-42)	14 (12-15)
20	24 (21-29)	21 (17-24)
15	16 (13-20)	31 (25-38)
10	11 (8-14)	45 (36-62)

*Data are means with 95% confidence intervals.

Other data including HCA at 10°C, report an increase in early and late neurocognitive and motor dysfunction when HCA exceeded 25 minutes.[19] More recent data including 490 consecutive patients undergoing HCA to a systemic or core temperature of 18-20°C coupled with topical cooling of the head, reported a significant difference in stroke rate of 0% for < 20 minutes, 0.7% for < 30 minutes, 1.3% for HCA < 50 minutes compared to 16.7% for HCA > 50 minutes.[20] Only hypothermic circulatory arrest of > 50 minutes was statistically different.[20]

The consensus on hypothermia classification on aortic surgery defines DHCA as a temperature between 14.1-20°C and MHCA as between 20.1-28°C.[21] (TABLE 2)

Category	Temperature (°C)
Profound	< 14
Deep	14.1-20
Low-moderate hypothermia	20.1-24
High-moderate hypothermia	24.1-28
Mild	28.1-34

Although there is no definitive 'safe' circulatory arrest time. When considering neurocognitive and motor functions, approximate 'safe' circulatory arrest times range from 30-40 minutes for profound hypothermia, 20-30 minutes for deep hypothermia, and 10-20 minutes for moderate hypothermia.[22] However, there is significant variability in management including degree of hypothermia, duration of pre-arrest cooling, blood gas management (alpha-stat vs pH stat), and patient individual cerebral vascular variabilities. Variabilities in Q10 value are reported between studies and different levels of hypothermia. Hypothermia alone may not yield cerebral electrical silence. In one study only 60% of cases have EEG silence at a core temperature of 18°C.[23] At a core temperature of 14-15°C the CMRO2 did not drop below 15% of the baseline.[16]

The effects of hypothermia depend on homogenous cooling of the end organs. For neurologic tissues predictors of electrical silence are temps < 12.5°C and cooling for > 50 minutes prior to circulatory arrest.[23] A longer duration of cooling at the desired temperature improves overall tissue protection.[22]

Risks of hypothermia

The benefits of hypothermia during circulatory arrest are balanced with the impact on other organ systems including, but not limited to the effects on coagulation, the heart and lungs, the kidneys, the splanchnic system, and peripheral muscle, the latter two being a large source of lactate buildup. While reduction in oxygen consumption and reduced need for high energy phosphates are of benefit, hypothermia also causes constriction, increased viscosity and sludging within the vascular tissues.[24] Solid organ dysfunctions are related to hypothermia induced vascular constriction and hypoperfusion.[6,11,24,25] The reduction in ATPase pump activity may increase intracellular sodium and calcium and cause cellular edema.24 Cold injury also impairs mitochondrial function.[24] Hypothermia negatively impacts on cardiac, pulmonary, splanchnic organs, and renal functions, and hematologic/coagulation functions. [3,6,10,11,25-30] A greater duration of hypothermia is directly related to bleeding, blood transfusion, end-organ dysfunction, and greater morbidity.[11,20,21,22,28,31,32]

Another 'risk' of hypothermia is rewarming, or, more specifically, the rate and degree of rewarming.[31] Rebound cerebral hyperthermia is potentially harmful and associated with postoperative neurocognitive dysfunction.[32,33] During the early phases of rewarming, jugular venous desaturation is suggestive of decreased oxygen delivery suggesting an imbalance between oxygen delivery and demand, the latter which is consistent with electroencephalographic reports of cerebral electrical hyperactivity during the rewarming phase.[32] In the early post CPB and postoperative periods mild degrees of hypothermia may be beneficial with regard to cerebral outcomes.

Adjuncts

To counter the adverse impact of circulatory arrest, perhaps increase the safety of prolonging it, adjunct techniques are included.[22] Adjuncts such as retrograde and antegrade cerebral perfusion improve cooling of the brain prior to the arrest and permit cerebral perfusion during circulatory arrest.[22,31] Retrograde cerebral perfusion (RCP) involves perfusion through the superior vena cava using a low pressure (< 20 mmHg), to promote better cerebral cooling and reduce the risk of cerebral ischemia. RCP can also help reduce arterial emboli. However, RCP with higher pressures (> 30 mmHg) may result in greater neurologic/neurocognitive dysfunction due to cerebral edema.[34] Anterograde cerebral profusion (ACP) was developed later and is now commonly used as an adjunct to perfuse the brain more physiologically with less risk of cerebral edema, and is preferred to retrograde cerebral perfusion.[22] Unilateral or bilateral ACP has been shown to improve survival for aortic arch repair procedures.[22,31] More recently an endo-aortic balloon technique has been introduced to establish normothermic perfusion to the rest of the body [35]

The use of hypothermia to slow metabolism increases the amount of time for the surgeon to perform complex procedures during circulatory arrest. The following 'pro/pro' discussion compares deep and moderate hypothermia during circulatory arrest.

References

1. Byrne JG, Aranki SF, Cohn LH. Aortic valve operations under deep hypothermic circulatory arrest for the porcelain aorta: "no-touch" technique. Ann Thorac Surg. 1998 May;65(5):1313-5. doi: 10.1016/s0003-4975(98)00183-0. PMID: 9594858.
2. Meling TR, Lavé A. What are the options for cardiac standstill during aneurysm surgery? A systematic review. Neurosurg Rev. 2019 Dec;42(4):843-852. doi: 10.1007/s10143-019-01183-4. Epub 2019 Oct 15. PMID: 31617125.
3. Clarke D. Circulation and Energy Metabolism of the Brain. In: Basic Neurochemistry: Molecular, Cellular and Medical Aspects.Vol Chapter 31. 6th ed. Philadelphia: Lippincott-Raven. https://www.ncbi.nlm.nih.gov/books/NBK20413/.
4. Erecinska M, Thoresen M, Silver IA. Effects of hypothermia on energy metabolism in Mammalian central nervous system. J Cereb Blood Flow Metab. 2003 May;23(5):513-30. doi: 10.1097/01.WCB.0000066287.21705.21. PMID: 12771566.
5. González-Ibarra FP, Varon J, López-Meza EG. Therapeutic hypothermia: critical review of the molecular mechanisms of action. Front Neurol. 2011 Feb 3;2:4. doi: 10.3389/fneur.2011.00004. PMID: 21331282; PMCID: PMC3035015.
6. Sandroni C, Cronberg T, Sekhon M. Brain injury after cardiac arrest: pathophysiology, treatment, and prognosis. Intensive Care Med. 2021 Dec;47(12):1393-1414. doi: 10.1007/s00134-021-06548-2. Epub 2021 Oct 27. PMID: 34705079; PMCID: PMC8548866.
7. Peterson MD, Garg V, Mazer CD, Chu MWA, Bozinovski J, Dagenais F, MacArthur RGG, Ouzounian M, Quan A, Jüni P, Bhatt DL, Marotta TR, Dickson J, Teoh H, Zuo F, Smith EE, Verma S, ACE CardioLinkκ 3 Trial Working Group Journal of thoracic and cardiovascular surgery, 2022, 164(5), 1426κ1438.e2 | added to CENTRAL: 28 February 2021 | 2021 Issue 02. https://doi.org/10.1016/j.jtcvs.2020.10.152
8. McCullough JN, Zhang N, Reich DL, Juvonen TS, Klein JJ, Spielvogel D, Ergin MA, Griepp RB. Cerebral metabolic suppression during hypothermic circulatory arrest in humans. Ann Thorac Surg. 1999 Jun;67(6):1895-9; discussion 1919-21. doi: 10.1016/s0003-4975(99)00441-5. PMID: 10391334.
9. Goldstein LJ, Davies RR, Rizzo JA, Davila JJ, Cooperberg MR, Shaw RK, Kopf GS, Elefteriades JA. Stroke in surgery of the thoracic aorta: incidence, impact, etiology, and prevention. J Thorac Cardiovasc Surg. 2001 Nov;122(5):935-45. doi: 10.1067/mtc.2001.117276. PMID: 11689799.
10. Itagaki S, Chikwe J, Sun E, Chu D, Toyoda N, Egorova N. Impact of Cerebral Perfusion on Outcomes of Aortic Surgery: The Society of Thoracic Surgeons Adult Cardiac Surgery Database Analysis. Ann Thorac Surg. 2020 Feb;109(2):428-435. doi: 10.1016/j.athoracsur.2019.08.043. Epub 2019 Sep 26. PMID: 31563489.
11 Qu JZ, Kao LW, Smith JE, Kuo A, Xue A, Iyer MH, Essandoh MK, Dalia AA. Brain Protection in Aortic Arch Surgery: An Evolving Field. J Cardiothorac Vasc Anesth. 2021 Apr;35(4):1176-1188. doi: 10.1053/j.jvca.2020.11.035. Epub 2020 Nov 21. PMID: 33309497.
12 Kirklin JW, Barrett-Boyes BG. Cardiac Surgery : Morphology, Diagnostic Criteria, Natural History, Techniques, Results, and Indications. New York: Churchill Livingstone, 1992.
13 Lewis FJ, Taufic M. Closure of atrial septal defects with the aid of hypothermia; experimental accomplishments and the report of one successful case. Surgery. 1953 Jan;33(1):52-9. PMID: 13015312.
14 Griepp RB, Stinson EB, Hollingsworth JF, Buehler D. Prosthetic replacement of the aortic arch. J Thorac Cardiovasc Surg. 1975 Dec;70(6):1051-63. PMID: 1186283.
15 McCullough JN, Zhang N, Reich DL, Juvonen TS, Klein JJ, Spielvogel D, Ergin MA, Griepp RB. Cerebral metabolic suppression during hypothermic circulatory arrest in humans. Ann Thorac Surg. 1999 Jun;67(6):1895-9; discussion 1919-21. doi: 10.1016/s0003-4975(99)00441-5. PMID: 10391334.

16 Erecinska M, Thoresen M, Silver IA. Effects of hypothermia on energy metabolism in Mammalian central nervous system. J Cereb Blood Flow Metab. 2003 May;23(5):513-30. doi: 10.1097/01.WCB.0000066287.21705.21. PMID: 12771566.

17 Haneda K, Sands MP, Thomas R, Hessel EA 2nd, Dillard DH. Prolongation of the safe interval of hypothermic circulatory arrest: 90 minutes. J Cardiovasc Surg (Torino). 1983 Jan-Feb;24(1):15-21. PMID: 6833347.

18 Tisherman SA, Safar P, Radovsky A, Peitzman A, Marrone G, Kuboyama K, Weinrauch V. Profound hypothermia (less than 10 degrees C) compared with deep hypothermia (15 degrees C) improves neurologic outcome in dogs after two hours' circulatory arrest induced to enable resuscitative surgery. J Trauma. 1991 Aug;31(8):1051-61; discussion 1061-2. PMID: 1875431.

19 Reich DL, Uysal S, Sliwinski M, Ergin MA, Kahn RA, Konstadt SN, McCullough J, Hibbard MR, Gordon WA, Griepp RB. Neuropsychologic outcome after deep hypothermic circulatory arrest in adults. J Thorac Cardiovasc Surg. 1999 Jan;117(1):156-63. doi: 10.1016/s0022-5223(99)70481-2. PMID: 9869770.

20 Ziganshin BA, Elefteriades JA. Deep hypothermic circulatory arrest. Ann Cardiothorac Surg. 2013 May;2(3):303-15. doi: 10.3978/j.issn.2225-319X.2013.01.05. PMID: 23977599; PMCID: PMC3741856.

21 Authors/Task Force Members; Czerny M, Grabenwöger M, Berger T, Aboyans V, Della Corte A, Chen EP, Desai ND, Dumfarth J, Elefteriades JA, Etz CD, Kim KM, Kreibich M, Lescan M, Di Marco L, Martens A, Mestres CA, Milojevic M, Nienaber CA, Piffaretti G, Preventza O, Quintana E, Rylski B, Schlett CL, Schoenhoff F, Trimarchi S, Tsagakis K; EACTS/STS Scientific Document Group; Siepe M, Estrera AL, Bavaria JE, Pacini D, Okita Y, Evangelista A, Harrington KB, Kachroo P, Hughes GC. EACTS/STS Guidelines for Diagnosing and Treating Acute and Chronic Syndromes of the Aortic Organ. Ann Thorac Surg. 2024 Jul;118(1):5-115. doi: 10.1016/j.athoracsur.2024.01.021. Epub 2024 Feb 26. PMID: 38416090.

22 Gutsche JT, Ghadimi K, Patel PA, Robinson AR 3rd, Lane BJ, Szeto WY, Augoustides JG. New frontiers in aortic therapy: focus on deep hypothermic circulatory arrest. J Cardiothorac Vasc Anesth. 2014 Aug;28(4):1159-63. doi: 10.1053/j.jvca.2014.03.018. PMID: 25107725.

23 Stecker MM, Cheung AT, Pochettino A, Kent GP, Patterson T, Weiss SJ, Bavaria JE. Deep hypothermic circulatory arrest: I. Effects of cooling on electroencephalogram and evoked potentials. Ann Thorac Surg. 2001 Jan;71(1):14-21. doi: 10.1016/s0003-4975(00)01592-7. PMID: 11216734.

24 Tveita T, Sieck GC. Physiological Impact of Hypothermia: The Good, the Bad, and the Ugly. Physiology (Bethesda). 2022 Mar 1;37(2):69-87. doi: 10.1152/physiol.00025.2021. Epub 2021 Oct 11. PMID: 34632808.

25 Svensson LG, Crawford ES, Hess KR, Coselli JS, Raskin S, Shenaq SA, Safi HJ. Deep hypothermia with circulatory arrest. Determinants of stroke and early mortality in 656 patients. J Thorac Cardiovasc Surg. 1993 Jul;106(1):19-28; discussion 28-31. PMID: 8321002.

26 Gega A, Rizzo JA, Johnson MH, Tranquilli M, Farkas EA, Elefteriades JA. Straight deep hypothermic arrest: experience in 394 patients supports its effectiveness as a sole means of brain preservation. Ann Thorac Surg. 2007 Sep;84(3):759-66; discussion 766-7. doi: 10.1016/j.athoracsur.2007.04.107. PMID: 17720372.

27 Sabharwal R, Johns EJ, Egginton S. The influence of acute hypothermia on renal function of anaesthetized euthermic and acclimatized rats. Exp Physiol. 2004 Jul;89(4):455-63. doi: 10.1113/expphysiol.2004.027904. Epub 2004 May 6. PMID: 15131076.

28 Khaladj N, Peterss S, Pichlmaier M, Shrestha M, von Wasielewski R, Hoy L, Haverich A, Hagl C. The impact of deep and moderate body temperatures on end-organ function during hypothermic circulatory arrest. Eur J Cardiothorac Surg. 2011 Dec;40(6):1492-9; discussion 1499. doi: 10.1016/j.ejcts.2011.03.031. Epub 2011 Apr 30. PMID: 21531569.

29. Rajagopalan S, Mascha E, Na J, Sessler DI. The effects of mild perioperative hypothermia on blood loss and transfusion requirement. Anesthesiology. 2008 Jan;108(1):71-7. doi: 10.1097/01.anes.0000296719.73450.52. PMID: 18156884.

30 Wolberg AS, Meng ZH, Monroe DM 3rd, Hoffman M. A systematic evaluation of the effect of temperature on coagulation enzyme activity and platelet function. J Trauma. 2004 Jun;56(6):1221-8. doi: 10.1097/01.ta.0000064328.97941.fc. PMID: 15211129.

31 Ziganshin BA, Elefteriades JA. Deep hypothermic circulatory arrest. Ann Cardiothorac Surg. 2013 May;2(3):303-15. doi: 10.3978/j.issn.2225-319X.2013.01.05. PMID: 23977599; PMCID: PMC3741856.

32 Svyatets M, Tolani K, Zhang M, Tulman G, Charchaflieh J. Perioperative management of deep hypothermic circulatory arrest. J Cardiothorac Vasc Anesth. 2010 Aug;24(4):644-55. doi: 10.1053/j.jvca.2010.02.010. Epub 2010 May 15. PMID: 20472472.

33 Conolly S, Arrowsmith JE, Klein AA: Deep hypothermic circulatory arrest. Continuing Education in Anaesthesia Critical Care & Pain 2010;10 (5). 138-142. https://doi.org/10.1093/bjace

34 Ueda Y. A reappraisal of retrograde cerebral perfusion. Ann Cardiothorac Surg. 2013 May;2(3):316-25. doi: 10.3978/j.issn.2225-319X.2013.01.02. PMID: 23977600; PMCID: PMC3741850.

35 Malvindi PG, Alfonsi J, Berretta P, Cefarelli M, Gatta E, Di Eusanio M. Normothermic frozen elephant trunk: our experience and literature review. Cardiovasc Diagn Ther. 2022 Jun;12(3):262-271. doi: 10.21037/cdt-22-73. PMID: 35800357; PMCID: PMC9253169.

Deep Hypothermic Circulatory Arrest for Aortic Arch Surgery: Less Complexity Locally, More Protection Distally

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Introduction

Deep hypothermic circulatory arrest (DHCA) has served as the cornerstone of cerebral and systemic protection during complex aortic arch repair with origins in the 1950's and 1960's.[1] By lowering core temperature to ≤ 20 °C, DHCA achieves near-electrocerebral silence and profound metabolic suppression, enabling a bloodless operative field and protecting the brain and distal organs. Although moderate hypothermia with adjuncts, including antegrade cerebral perfusion (ACP) and retrograde cerebral perfusion (RCP), has gained popularity, its putative benefits rest primarily on retrospective analyses inherent with important confounding factors. We argue that contemporary physiological evidence and randomized data reaffirm that DHCA remains the simplest, most reproducible, and safest global protection strategy for circulatory arrest.

Limitations of Evidence Favoring Moderate Hypothermia

The enthusiasm for moderate hypothermia has been driven largely by retrospective, observational series with heterogeneous patient populations, inconsistent temperature definitions, and variable perfusion strategies. Practice variation between institutions and within pooled retrospective clinical data leads to difficulty isolating temperature as an independent determinant of outcome. Major confounders include arrest duration, selective cerebral perfusion strategies, monitoring sites, surgical technique, complexity of the repair, rate of cooling and rewarming, and baseline neurologic risk.[2,3]

Augoustides et al. emphasized that the question is not simply "deep versus moderate" but a complex interplay of metabolic milieu, pH management, and perfusion technique, of which none are standardized across institutions.4 Similarly, Qu et al. noted that published results of moderate hypothermia must be interpreted within the context of uncontrolled variability in cerebral monitoring and incomplete reporting of distal organ outcomes.[5] The 2022 ACC/AHA aortic disease guidelines accordingly state that no single temperature or perfusion strategy has been shown conclusively to be superior for all patients.6

Thus, the purported equivalence of outcomes in moderate hypothermia series cannot be separated from patient selection and institutional expertise. Only a rigorously conducted randomized trial can clarify whether lesser hypothermia matches DHCA in neurologic and systemic protection.

Randomized Data: The GOT ICE Trial

The *Cognitive Effects of Body Temperature During Hypothermic Circulatory Arrest (*GOT ICE) trial is the first multicenter randomized comparison of deep, low-moderate, and high-moderate hypothermia, all with the use of unilateral ACP.[7] Across 282 patients, no difference in global cognitive change or major morbidity was observed among groups. Importantly, structured verbal memory was better preserved in the deep group compared with high-moderate hypothermia (P = 0.036).[7] While transfusion volume was numerically greater with deep cooling, this did not translate into higher rates of re-exploration, infection, or prolonged ventilation, leaving the clinical significance of this finding questionable. Operative times and overall complication rates were also equivalent.[7] These data reinforce that DHCA is at least equivalent, and possibly advantageous, in neurocognitive outcomes without excess morbidity.

Neurologic Protection Under Deep Hypothermia

Profound hypothermia decreases cerebral metabolic rate by approximately 6–7 % per °C, achieving >80 % suppression at 18 °C.[8] This predictable metabolic quiescence alone can allow up to 30–40 minutes of safe global ischemia. Luehr et al. caution that as body temperatures rise, the "safe" arrest window shortens precipitously, exposing the brain to under-recognized

ischemic stress.[8] Employment of moderate hypothermia relies on continuous ACP to offset this vulnerability, yet cerebral flow distribution during unilateral ACP is non-uniform and difficult to monitor reliably.

Near-infrared spectroscopy (NIRS) provides only limited cortical information, and discrepancies between hemispheres may go undetected.[5,8] Experimental data reveal substantial inter-individual variation in the anatomy of the circle of Willis completeness; hence, unilateral ACP risks regional hypoperfusion despite apparently adequate flow. Luehr et al. and Elefteriades et al. both highlight that deep hypothermia obviates these uncertainties by ensuring uniform metabolic protection independent of vascular anatomy or monitoring limitations.[8,10]

Safety and Simplicity of the DHCA Technique

As Elefteriades et al. describe, "straight" DHCA—deep hypothermia without adjunct perfusion—provides a simple, reproducible, and uncluttered operative field.[10] It avoids the technical hazards of cannulating fragile supra-aortic vessels and the uncertainty of determining ideal ACP flow rates. In a 10-year series of nearly 400 patients, Elefteriades reported a mean arrest time of 31 minutes, stroke rate 2.3 %, and mortality 2.2 % using this technique.[10] The avoidance of additional tubing and cannulas also shortens setup time and minimizes embolic risk.

Conversely, ACP and RCP introduce potential for cannula-related trauma, atheroembolic stroke, and malperfusion from incorrect flow distribution. There is no consensus regarding how many head vessels should be perfused or at what rates, with both hypoperfusion and cerebral edema possible if flow is misjudged.[11] These uncertainties are eliminated under straight DHCA.

Coagulation and Bleeding

Concerns regarding coagulopathy under deep hypothermia may be overstated. Harrington et al. found that profound hypothermia was not an independent predictor of postoperative bleeding, renal dysfunction, or prolonged stay in the intensive care unit.[12] Multivariate analysis instead identified cardiopulmonary-bypass time and procedure extent as the primary drivers of hemorrhage and organ dysfunction. The authors concluded that "procedure extent, not temperature, determines postoperative bleeding".[12] Another study described over 700 patients and found no difference in reoperation for bleeding between profound and deep hypothermia groups.[8] These findings mimic the GOT ICE observation that the modest increase in transfusion volume under DHCA lacks clinical consequence.[7]

While hypothermia alters platelet function and enzymatic coagulation, the effect is likely clinically minor when modern perfusion management, antifibrinolytics, and meticulous rewarming are used.

Renal and Visceral Organ Protection

Systemic organ protection beyond the brain is another consideration. Moderate hypothermia with ACP preferentially perfuses the head, leaving the spinal cord, kidneys, and viscera ischemic at relatively warmer temperatures. The spinal cord and kidneys specifically exhibit low tolerance for ischemia and may suffer subclinical injury. Cao et al.'s meta-analysis of 14 observational studies (4,142 patients) found a lower rate of renal failure with moderate hypothermia overall (OR 0.76; 95% CI 0.61–0.94) but no significant difference when circulatory arrest exceeded 30 minutes.[13] Because most complex arch repairs require longer arrests and unintended prolonged arrest times may occur, DHCA provides equal renal protection in the scenarios where protection is most needed. It is also important to consider that all studies included in the meta-analysis were retrospective and at risk of selection bias, with confidence intervals crossing 1.[13]

Animal models demonstrate that the spinal cord and visceral organs tolerate arrest poorly at moderate temperatures. Luehr et al. summarized both experimental and clinical data showing that "trading effective neuroprotection for warm distal ischemia constitutes a significant step backward," jeopardizing spinal and visceral integrity.[8] Etz et al further reported spinal cord ischemia as a function of distal temperature, with irreversible injury occurring earlier under moderate hypothermia.[9] These observations underscore the whole-body safety margin intrinsic to DHCA.

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Technical and Physiologic Variability of Moderate Hypothermia

It is critical that circulatory arrest performed under moderate hypothermia employs adjuncts such as ACP or RCP. Moderate hypothermia with ACP introduces multiple sources of error: choice of cannulation site (right axillary, innominate, or carotid), unilateral versus bilateral perfusion, variable flow rates (5–15 mL/kg/min), and differing perfusate temperatures (12–20 °C). Each adjustment influences cerebral hemodynamics and metabolic suppression. One must take caution in interpreting evidence supporting moderate hypothermia with ACP given that no standardized approach exists.8 Even subtle miscalibration can result in asymmetric perfusion or excessive flows, resulting in cerebral edema. Moreover, the assumption of an intact circle of Willis is often false, rendering unilateral ACP potentially unreliable.[8]

In contrast, DHCA requires no manipulation of the supra-aortic trunks, avoids potential embolic sources, and maintains uniform protection irrespective of vascular variation. For these reasons, DHCA remains the most forgiving and reproducible technique across diverse surgical and institutional settings.

Conclusion

Deep hypothermic circulatory arrest remains the most physiologically grounded, reliable method for achieving global organ protection during aortic arch surgery. Although modern adjuncts such as ACP and RCP have expanded the safe operative window, the foundation of protection continues to be deep systemic cooling. Retrospective studies supporting moderate hypothermia are limited by a high degree of confounding factors. Randomized, prospective data demonstrate at best equivalence—and possible neurocognitive superiority—for deep hypothermia. Concerns about coagulopathy have not translated into important patient outcomes in the same high-level evidence. Further, deep hypothermia provides a wider safety margin for renal protection.

In an operating room where the margin for error is measured in millimeters, simplicity and predictability are foundational. DHCA offers both reliable cerebral protection, inherent safety for the spinal cord and viscera, and a streamlined operative workflow. Deep hypothermia remains the evidence-based option for patient safety during circulatory arrest.

References

1. Rimmer L, Fok M, Bashir M. The History of Deep Hypothermic Circulatory Arrest in Thoracic Aortic Surgery. Aorta (Stamford). 2014;2(4):129-134. Published 2014 Aug 1. doi:10.12945/j.aorta.2014.13-049
2. Englum BR, Andersen ND, Husain AM, Mathew JP, Hughes GC. Degree of hypothermia in aortic arch surgery - optimal temperature for cerebral and spinal protection: deep hypothermia remains the gold standard in the absence of randomized data. Ann Cardiothorac Surg. 2013;2(2):184-193. doi:10.3978/j.issn.2225-319X.2013.03.01
3. Harky A, Bashir M, Mariscalco G. Aortic arch aneurysm surgery: what is the gold standard temperature in the absence of randomized data?. Gen Thorac Cardiovasc Surg. 2019;67(1):127-131. doi:10.1007/s11748-017-0867-9
4. Augoustides JG. What are the clinical questions for optimal conduct of deep hypothermic circulatory arrest for adult aortic arch repair?. J Cardiothorac Vasc Anesth. 2007;21(6):918-919. doi:10.1053/j.jvca.2006.12.018
5. Qu JZ, Kao LW, Smith JE, et al. Brain Protection in Aortic Arch Surgery: An Evolving Field. J Cardiothorac Vasc Anesth. 2021;35(4):1176-1188. doi:10.1053/j.jvca.2020.11.035
6. Isselbacher EM, Preventza O, Hamilton Black J 3rd, et al. 2022 ACC/AHA Guideline for the Diagnosis and Management of Aortic Disease: A Report of the American Heart Association/American College of Cardiology Joint Committee on Clinical Practice Guidelines. Circulation. 2022;146(24):e334-e482. doi:10.1161/CIR.0000000000001106
7. Hughes GC, Chen EP, Browndyke JN, et al. Cognitive Effects of Body Temperature During Hypothermic Circulatory Arrest Trial (GOT ICE): A Randomized Clinical Trial Comparing Outcomes After Aortic Arch Surgery. Circulation. 2024;149(9):658-668. doi:10.1161/

CIRCULATIONAHA.123.067022

8. Luehr M, Bachet J, Mohr FW, Etz CD. Modern temperature management in aortic arch surgery: the dilemma of moderate hypothermia. Eur J Cardiothorac Surg. 2014;45(1):27-39. doi:10.1093/ejcts/ezt154.
9. Etz CD, Luehr M, Kari FA, et al. Selective cerebral perfusion at 28 degrees C--is the spinal cord safe?. Eur J Cardiothorac Surg. 2009;36(6):946-955. doi:10.1016/j.ejcts.2009.05.046
10. Elefteriades JA. What is the best method for brain protection in surgery of the aortic arch? Straight DHCA. Cardiol Clin. 2010;28(2):381-387. doi:10.1016/j.ccl.2010.02.004.
11. Ziganshin BA, Elefteriades JA. Deep hypothermic circulatory arrest. Ann Cardiothorac Surg. 2013;2(3):303-315. doi:10.3978/j.issn.2225-319X.2013.01.05
12. Harrington DK, Lilley JP, Rooney SJ, Bonser RS. Nonneurologic morbidity and profound hypothermia in aortic surgery. Ann Thorac Surg. 2004;78(2):596-601. doi:10.1016/j.athoracsur.2004.01.012
13. Cao L, Guo X, Jia Y, Yang L, Wang H, Yuan S. Effect of Deep Hypothermic Circulatory Arrest Versus Moderate Hypothermic Circulatory Arrest in Aortic Arch Surgery on Postoperative Renal Function: A Systematic Review and Meta-Analysis. J Am Heart Assoc. 2020;9(19):e017939. doi:10.1161/JAHA.120.017939

Moderate Hypothermia Less Bleeding, Faster Rewarm, Same Brain for Circulatory Arrest

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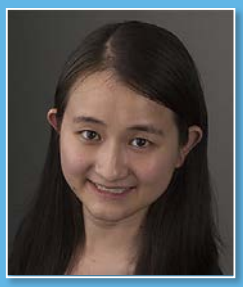

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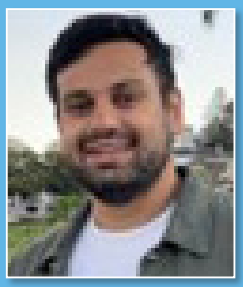

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Introduction

Therapeutic hypothermia is an important method of neuroprotection and is used in neurological disorders as well as after return of spontaneous circulation (ROSC) following cardiac arrest to improve survival and limit neurologic injury. Several mechanisms have been proposed to explain how hypothermia improves neurological outcomes, including a reduction in electrical activity and a decrease in the cerebral metabolic rate for oxygen ($CMRO_2$). Despite these potential benefits, hypothermia can also produce adverse effects, such as infection and coagulopathy.

In this discussion, we will review the reasons why moderate hypothermia during circulatory arrest offers overall advantages compared to deep hypothermia.

1. Definitions

Aortic arch repair under circulatory arrest is among the more demanding operations in both adult and pediatric cardiac surgery, performed for aneurysm, dissection, and selected congenital lesions. Hypothermic circulatory arrest remains the central organ-protection strategy because cooling lowers cerebral and systemic metabolic demand and thereby lengthens the tolerable ischemic interval. In current practice, two temperature ranges are used most often: deep hypothermia (roughly 14–20°C) and moderate hypothermia (about 20.1–28°C), with some centers extending moderate hypothermia up to 32°C. STS/EACTS guidelines further refine this into low-moderate (20.1–24°C), high-moderate (24.1–28°C), and mild hypothermia (28.1–34°C).The selected temperature almost always goes hand in hand with the cerebral protection strategy—typically antegrade cerebral perfusion (ACP) and, less commonly, retrograde cerebral perfusion (RCP)—which are now standard adjuncts.[1]

The move from traditional deep hypothermia to more moderate cooling has been driven by the desire to lessen penalties of profound hypothermia—coagulopathy, longer bypass times due to rewarming, and end-organ dysfunction—without sacrificing neurologic safety. In this review, we summarize contemporary data comparing moderate and deep hypothermic protocols for arch surgery with circulatory arrest, with attention to coagulation outcomes, rewarming duration, neurologic injury, procedure-related complications, and resource use in both adults and children.[1]

2. Comparative Outcomes: Moderate vs Deep Hypothermia

2.1 Coagulopathy and Bleeding

Coagulation impairment with deeper levels of hypothermia is a consistent finding in arch surgery. At very low temperatures, platelet adhesion and aggregation decline, coagulation-factor kinetics slow, and fibrinolysis is amplified; all of this sits on top of the hemodilution and CPB-related inflammatory response that already push patients toward bleeding.[1-3]

Keenan et al. analyzed 310 propensity-matched adults undergoing hemiarch replacement and showed that patients cooled only to the moderate range (mean 24.1°C) received less plasma (median 5 vs 6 units, $p = .01$), had slightly lower cell-saver return (472 vs 500 mL, $p < .01$), and bled less in the first 12 hours (350 vs 440 mL, $p < .01$) than those managed with deep hypothermia. Despite these differences, re-exploration for bleeding and global morbidity/mortality were unchanged.4 Poon et al. and Abjigitova et al. reached a similar conclusion: moderating the temperature reduces transfusion and drainage volumes, but the effect size is small and does not reliably shift hard outcomes such as reoperation.[5,6]

Pediatric data points in the same direction. Xie et al. compared children managed with moderate hypothermia plus selective ACP (mean 26.0°C) with those cooled to a deep level (mean 18.9°C) and found less 24-hour chest drainage (28.9 vs 47.4 mL/kg, $p < 0.05$), as well as shorter bypass runs, ventilation, and ICU stay in the moderately cooled group.[7]

These observations are in line with the EACTS/STS guidance, which notes that deep hypothermia

is more frequently accompanied by coagulopathy and a stronger systemic inflammatory response, and that moving toward moderate hypothermia with cerebral perfusion lessens these risks.1 Preventza et al. further reported that, in patients requiring longer cerebral-protection times, moderate hypothermia with ACP was associated with fewer reoperations for bleeding and better long-term survival than deep hypothermia.[8]

In summary, moderate hypothermia is associated with a modest but statistically significant reduction in coagulopathy and transfusion requirements compared to deep hypothermia (≤20°C) in both adults and children. The reduction in bleeding is most consistently observed as lower plasma transfusion volumes and chest tube output but does not always result in lower reoperation rates for bleeding or improved overall mortality.[1,4-8]

2.2 Rewarming Time and Operative Efficiency

Rewarming is one of the rate-limiting steps in arch surgery and often dictates both total pump time and overall operative duration. When patients are cooled to deep hypothermic levels, more time is needed not only to reach the target temperature but also to come back up safely, because fast rewarming from very low temperatures can produce cerebral hyperthermia and increase the risk of neurologic injury. By contrast, when arrest is done in the moderate range, rewarming can proceed faster and in a more physiologic fashion.[9]

In a cohort of 3,265 adults undergoing total arch replacement, Keeling et al. showed that patients managed with moderate hypothermia (approximately 20–28°C) had markedly shorter CPB times than those cooled to deep levels (12–20°C): 200 minutes vs 243 minutes ($p < 0.001$).9 Most of that 43-minute gap was explained by shorter cooling and rewarming phases. Similar conclusions were reported by Poon et al. and are reflected in the EACTS/STS guidelines: moderating the temperature shortens the case without paying a penalty in mortality or major morbidity.[1,5]

Pediatric data are consistent. Xie et al. found that children treated with moderate hypothermia plus ACP had shorter CPB times (146.9 ± 40.6 min) than those cooled to deep hypothermia (189.6 ± 41.2 min, $p < 0.05$). Even though rewarming was not broken out separately, the shorter pump run is an indirect marker of less time spent returning to normothermia.[7]

In practical terms, emerging from deep hypothermia often takes 40–60 minutes, whereas rewarming from a moderate target can be completed in about 20–30 minutes, depending on patient size and institutional protocols. Time saved is not just a convenience; it reduces exposure to CPB-related coagulopathy, end-organ stress, and the risk of overshooting brain temperature.[1,5,10]

In summary, moderate hypothermia protocols are associated with a reduction in total CPB time compared to deep hypothermia, with the majority of this difference due to shorter rewarming. This translates into reduced operative risk and improved perioperative efficiency in both adult and pediatric populations.[1,5,7,10]

2.3 Neurologic Outcomes

Neurologic injury remains the most worrisome adverse outcome in arch surgery with circulatory arrest—stroke, early neurocognitive decline, and longer-term structural brain changes are ultimately what determine whether a technically successful operation is a good operation. The available data come from several layers of evidence: randomized trials, large registry analyses, and multiple meta-analyses.

The EACTS/STS document, which pulls together those data, notes that using moderate hypothermia together with ACP is associated with lower operative mortality and a lower risk of postoperative stroke than traditional deep hypothermia.1 A network meta-analysis including 12,370 patients showed the same signal: patients cooled to deep hypothermia had more strokes than those managed with moderate hypothermia (OR 1.46, 95% CI 1.19–1.78).[6] In the Canadian Thoracic Aortic Collaborative registry (2,520 patients), a nadir temperature ≥24°C during circulatory arrest predicted better survival and fewer strokes than cooler strategies, and use of ACP added further protection.[11]

Prospective data from the GOT ICE trial are especially helpful. In that study, 282 adults

were randomized to deep (≤20°C), low-moderate (20.1–24°C), or high-moderate (24.1–28°C) hypothermia during HCA with ACP. Global cognitive performance at 4 weeks was no worse with low- or high-moderate hypothermia than with deep hypothermia, meeting non-inferiority. A domain analysis did show slightly better structured verbal memory in the deep group, but this was a secondary finding and not accompanied by differences in mortality, major morbidity, or quality of life. Notably, all patients, regardless of target temperature, showed MRI evidence of reduced gray-matter volume and cortical thickness, underscoring that the injury risk is inherent to HCA itself.[12]

Longer-term follow-up favors the more moderate strategies as well. In 544 patients requiring ACP for more than 30 minutes, 4- and 8-year survival was higher after moderate hypothermia (75.4% and 74.2%) than after deep hypothermia (62.3% and 55.7%).8 A separate single-center series of 1,310 patients found that high-moderate hypothermia (24.1–28°C) was an independent protective factor for operative mortality, without a penalty in late survival or quality of life.[13]

Pediatric experience tracks closely with the adult data. Xie et al. reported fewer neurologic complications when children were managed with moderate hypothermia plus ACP than with deep hypothermia (4.9% vs 18.9%, $p < 0.05$).[7] Kornilov et al. saw a similar pattern in infants undergoing arch reconstruction—neurologic events were markedly less frequent with moderate hypothermia and ACP (5.9% vs 30.8%, $p = 0.02$).[14]

In summary, moderate hypothermia, particularly when combined with ACP, is associated with lower rates of stroke and permanent neurologic dysfunction compared to deep hypothermia in both adult and pediatric patients. Neurocognitive outcomes are broadly similar between temperature strategies, with moderate hypothermia being noninferior to deep hypothermia for global cognitive function.[1,6-8,11-14]

3. Adverse Events and Resource Utilization

Recent meta-analyses and large single- and multicenter series have helped define the complication patterns seen with different hypothermia strategies in arch surgery. The events of greatest interest remain renal dysfunction, end-organ injury, infection, and arrhythmia.

Renal injury is the clearest signal. In a systematic review of 4,142 patients, moderate hypothermia was linked to a lower rate of postoperative renal failure (OR 0.76, 95% CI 0.61–0.94) and a reduced need for renal replacement therapy (OR 0.68, 95% CI 0.48–0.97) compared with deep hypothermia, with the advantage most evident when circulatory arrest was <30 minutes.[15] A larger network meta-analysis of 12,370 patients likewise found a higher AKI risk with deep hypothermia.[6] Given the shorter bypass times associated with rewarming—this pattern supports favoring moderate or low-moderate hypothermia in some observational studies.

For other postoperative complications the differences are less pronounced. Rates of infection and arrhythmia appear broadly similar between moderate and deep hypothermia in pooled data, and this is reflected in the EACTS/STS document, which does not make a temperature-specific recommendation on these endpoints.[1] What is more consistent is the observation that deeper cooling is accompanied by a stronger systemic inflammatory response, more coagulopathy, and a higher burden of end-organ dysfunction—hepatic, pulmonary, or both—providing part of the rationale for moving toward warmer strategies.[6,15-17]

Resource use also favors moderate hypothermia. Warmer protocols are associated with shorter cooling/rewarming phases and therefore shorter CPB and operative times, lower transfusion needs, and shorter ICU and hospital stays. Pediatric series show the same pattern: moderate hypothermia with ACP shortened bypass, reduced bleeding, decreased ICU time, and was accompanied by fewer neurologic events than deep hypothermia. Although formal cost analyses are limited, these efficiency gains almost certainly translate into better cost-effectiveness.[1,4,5,7,10,14]

In summary, moderate hypothermia is associated with a lower risk of renal dysfunction and end-organ injury, with no increase in infection or arrhythmia, and is likely to be more cost-effective than deep hypothermia protocols in both adults and children.[1,6,7,10,15-17]

CON

4. Guideline Recommendations and Implementation

Current international guidance from the STS, EACTS, AATS, and AHA has gradually shifted toward the use of moderate hypothermia with cerebral perfusion as the default approach for aortic arch surgery requiring circulatory arrest, in both adult and pediatric patients.[1,18,19]

The EACTS/STS document defines high-moderate hypothermia as 24.1–28°C and recommends this range, together with cerebral perfusion—preferably ACP—for most arch procedures. For hemiarch replacement, high-moderate hypothermia plus cerebral perfusion carries a Class I, Level C recommendation; for more complex arch reconstruction, high-moderate hypothermia with selective ACP is Class IIa, Level B.[1] The guideline further advises using both nasopharyngeal and core temperature monitoring, initiating ACP before circulatory arrest, keeping arrest times as short as possible, and performing slow, controlled rewarming to limit neurologic injury.[1,20]

The AATS similarly notes that, in acute type A dissection repair, moderate hypothermia used with ACP is safe and achieves outcomes at least comparable with deep hypothermia, and that current data do not clearly favor unilateral over bilateral perfusion.[18] The AHA concurs, highlighting better neurologic outcomes and lower mortality with moderate hypothermia plus cerebral perfusion than with deep hypothermia alone, particularly when longer arrest intervals are anticipated.[19]

For children, EACTS/STS recommends extrapolating the adult strategy—moderate hypothermia (typically 24–28°C) plus ACP—because the pathophysiologic rationale is the same and the pediatric series available to date show similar advantages in bleeding, neurologic events, and ICU stay. Individualization is still advised, considering patient size, arch complexity, and expected arrest time.[1,7,14]

In summary, the overarching message from these societies is consistent: moderate hypothermia combined with cerebral perfusion should be considered the preferred strategy for arch surgery with circulatory arrest, offering equal or better neurologic protection, less coagulopathy, and, in several series, improved survival compared with deep hypothermia.[1,18,19]

5. Limitations and Future Directions

Although current data strongly favor moderate hypothermia combined with cerebral perfusion for arch surgery, the evidence base is not without gaps—this is most obvious in children. Most pediatric reports are single- or multicenter retrospective series, inherently vulnerable to confounding, selection bias, and evolution of practice over time, and there are no pediatric randomized or prospective trials that directly compare moderate versus deep hypothermia for bleeding, rewarming, or neurologic endpoints. Thus, pediatric recommendations are largely inferred from adult experience and expert consensus rather than from level I pediatric data.[1,7,14]

A second limitation is inconsistency in how temperature ranges are defined and measured.[1,6,13] The use of adjunctive cerebral perfusion techniques is not uniform, and differences in perfusion strategy, flow rates, and cannulation techniques may confound the impact of temperature alone.5,21Advances in surgical technique, perfusion technology, and perioperative care over time may also confound the observed benefits of moderate hypothermia.[1,22]

Outcome reporting is another weak point. Retrospective studies may underreport complications or omit long-term follow-up, and neurocognitive outcomes are particularly heterogeneous—formal testing is not universal, follow-up is often short, and subtle deficits may be missed. Future work should therefore emphasize prospective pediatric cohorts, standardized temperature definitions, uniform cerebral perfusion protocols, and validated neurocognitive endpoints.[12,13,23]

Despite these limitations, the overall signal across guidelines, meta-analyses, and contemporary series is consistent: moderate hypothermia used with cerebral perfusion reduces coagulopathy, shortens cooling/rewarming and total CPB time, and provides neurologic protection that is at least equivalent—and in some analyses superior—to deep hypothermia. The final choice of target temperature should still be individualized to patient size, arch complexity, and anticipated arrest time, but current international recommendations place moderate hypothermia as the preferred strategy.

CON

References

1. Czerny M, Grabenwöger M, Berger T, et al. EACTS/STS Guidelines for Diagnosing and Treating Acute and Chronic Syndromes of the Aortic Organ. Ann Thorac Surg. Jul 2024;118(1):5-115. doi:10.1016/j.athoracsur.2024.01.021
2. Ghia S, Savadjian A, Shin D, Diluozzo G, Weiner MM, Bhatt HV. Hypothermic Circulatory Arrest in Adult Aortic Arch Surgery: A Review of Hypothermic Circulatory Arrest and its Anesthetic Implications. J Cardiothorac Vasc Anesth. Dec 2023;37(12):2634-2645. doi:10.1053/j.jvca.2023.08.139
3. Fernández Suárez FE, Fernández Del Valle D, González Alvarez A, Pérez-Lozano B. Intraoperative care for aortic surgery using circulatory arrest. J Thorac Dis. May 2017;9(Suppl 6):S508-S520. doi:10.21037/jtd.2017.04.67
4. Keenan JE, Wang H, Gulack BC, et al. Does moderate hypothermia really carry less bleeding risk than deep hypothermia for circulatory arrest? A propensity-matched comparison in hemiarch replacement. J Thorac Cardiovasc Surg. Dec 2016;152(6):1559-1569.e2. doi:10.1016/j.jtcvs.2016.08.014
5. Poon SS, Estrera A, Oo A, Field M. Is moderate hypothermic circulatory arrest with selective antegrade cerebral perfusion superior to deep hypothermic circulatory arrest in elective aortic arch surgery? Interact Cardiovasc Thorac Surg. Sep 2016;23(3):462-8. doi:10.1093/icvts/ivw124
6. Abjigitova D, Notenboom ML, Veen KM, et al. Optimal temperature management in aortic arch surgery: A systematic review and network meta-analysis. J Card Surg. Dec 2022;37(12):5379-5387. doi:10.1111/jocs.17206
7. Xie L, Xu Y, Huang G, et al. MHCA with SACP versus DHCA in Pediatric Aortic Arch Surgery: A Comparative Study. Sci Rep. Mar 10 2020;10(1):4439. doi:10.1038/s41598-020-61428-x
8. Preventza O, Coselli JS, Akvan S, et al. The impact of temperature in aortic arch surgery patients receiving antegrade cerebral perfusion for >30 minutes: How relevant is it really? J Thorac Cardiovasc Surg. Apr 2017;153(4):767-776. doi:10.1016/j.jtcvs.2016.11.059
9. Hiratzka LF, Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for the diagnosis and management of patients with thoracic aortic disease: Executive summary: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. Anesth Analg. Aug 2010;111(2):279-315. doi:10.1213/ANE.0b013e3181dd869b
10. Keeling WB, Tian DH, Leshnower BG, et al. Safety of Moderate Hypothermia With Antegrade Cerebral Perfusion in Total Aortic Arch Replacement. Ann Thorac Surg. Jan 2018;105(1):54-61. doi:10.1016/j.athoracsur.2017.06.072
11. Hage A, Stevens LM, Ouzounian M, et al. Impact of brain protection strategies on mortality and stroke in patients undergoing aortic arch repair with hypothermic circulatory arrest: evidence from the Canadian Thoracic Aortic Collaborative. Eur J Cardiothorac Surg. Jul 01 2020;58(1):95-103. doi:10.1093/ejcts/ezaa023
12. Hughes GC, Chen EP, Browndyke JN, et al. Cognitive Effects of Body Temperature During Hypothermic Circulatory Arrest Trial (GOT ICE): A Randomized Clinical Trial Comparing Outcomes After Aortic Arch Surgery. Circulation. Feb 27 2024;149(9):658-668. doi:10.1161/CIRCULATIONAHA.123.067022
13. Zhang K, Zhou C, Gao S, et al. The optimal degree of core temperature for hypothermic circulatory arrest in complex aortic arch surgery: results from 1310 patients. Eur J Cardiothorac Surg. Aug 02 2024;66(2)doi:10.1093/ejcts/ezae311

14. Kornilov IA, Sinelnikov YS, Soinov IA, et al. Outcomes after aortic arch reconstruction for infants: deep hypothermic circulatory arrest versus moderate hypothermia with selective antegrade cerebral perfusion. Eur J Cardiothorac Surg. Sep 2015;48(3):e45-50. doi:10.1093/ejcts/ezv235
15. Cao L, Guo X, Jia Y, Yang L, Wang H, Yuan S. Effect of Deep Hypothermic Circulatory Arrest Versus Moderate Hypothermic Circulatory Arrest in Aortic Arch Surgery on Postoperative Renal Function: A Systematic Review and Meta-Analysis. J Am Heart Assoc. Oct 20 2020;9(19):e017939. doi:10.1161/JAHA.120.017939
16. Arnaoutakis GJ, Vallabhajosyula P, Bavaria JE, et al. The Impact of Deep Versus Moderate Hypothermia on Postoperative Kidney Function After Elective Aortic Hemiarch Repair. Ann Thorac Surg. Oct 2016;102(4):1313-21. doi:10.1016/j.athoracsur.2016.04.007
17. Vekstein AM, Yerokun BA, Jawitz OK, et al. Does deeper hypothermia reduce the risk of acute kidney injury after circulatory arrest for aortic arch surgery? Eur J Cardiothorac Surg. Jul 30 2021;60(2):314-321. doi:10.1093/ejcts/ezab044
18. Malaisrie SC, Szeto WY, Halas M, et al. 2021 The American Association for Thoracic Surgery expert consensus document: Surgical treatment of acute type A aortic dissection. J Thorac Cardiovasc Surg. Sep 2021;162(3):735-758.e2. doi:10.1016/j.jtcvs.2021.04.053
19. Gaudino M, Benesch C, Bakaeen F, et al. Considerations for Reduction of Risk of Perioperative Stroke in Adult Patients Undergoing Cardiac and Thoracic Aortic Operations: A Scientific Statement From the American Heart Association. Circulation. Oct 06 2020;142(14):e193-e209. doi:10.1161/CIR.0000000000000885
20. Russo AM, Stainback RF, Bailey SR, et al. ACCF/HRS/AHA/ASE/HFSA/SCAI/SCCT/SCMR 2013 appropriate use criteria for implantable cardioverter-defibrillators and cardiac resynchronization therapy: a report of the American College of Cardiology Foundation appropriate use criteria task force, Heart Rhythm Society, American Heart Association, American Society of Echocardiography, Heart Failure Society of America, Society for Cardiovascular Angiography and Interventions, Society of Cardiovascular Computed Tomography, and Society for Cardiovascular Magnetic Resonance. J Am Coll Cardiol. Mar 26 2013;61(12):1318-68. doi:10.1016/j.jacc.2012.12.017
21. Leshnower BG, Rangaraju S, Allen JW, Stringer AY, Gleason TG, Chen EP. Deep Hypothermia With Retrograde Cerebral Perfusion Versus Moderate Hypothermia With Antegrade Cerebral Perfusion for Arch Surgery. Ann Thorac Surg. Apr 2019;107(4):1104-1110. doi:10.1016/j.athoracsur.2018.10.008
22. Qu JZ, Kao LW, Smith JE, et al. Brain Protection in Aortic Arch Surgery: An Evolving Field. J Cardiothorac Vasc Anesth. Apr 2021;35(4):1176-1188. doi:10.1053/j.jvca.2020.11.035
23. Centofanti P, Barbero C, D'Agata F, et al. Neurologic and cognitive outcomes after aortic arch operation with hypothermic circulatory arrest. Surgery. Sep 2016;160(3):796-804. doi:10.1016/j.surg.2016.02.008