SCA NEWS

SOCIETY OF CARDIOVASCULAR ANESTHESIOLOGISTS
Knowledge • Care • Investigation

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April 2022

PRESIDENT'S MESSAGE

Andrew Shaw
MB, FCCM, FFICM, FRCA
President, Society of Cardiovascular Anesthesiologists

"Education is the Lifeblood of our Society"

Reflections at the Halfway Marker

We will be holding our Annual Meeting and Workshops in Palm Springs, CA – a time at which we welcome newly elected Board and Committee members to our SCA volunteer community and charge our various teams with projects for the coming year.

This meeting also marks the halfway point for my tenure as SCA President and thus an opportunity to reflect on goals, aspirations, and our related achievements. In this newsletter message, I will review the year we have come through and look ahead to next year as we advance our plans and agenda for the Society's activities.

Last April, we held an entirely virtual Annual Meeting. Against the odds, our extraordinary Scientific Planning Committee, led by Drs. Sasha Shillcutt, Mary-Beth Brady, and Jonathan Ho accomplished a fantastic feat and delivered an incredibly successful meeting. The feedback we received from members was universally positive, in some cases excellent.

We should publicly recognize our Scientific Planning Committee for their hard work and incredible results they continue to deliver, year in and year out. We have become so used to the high quality of our educational events that we might be thinking it's easy to work and anyone can do it.

Of course, it is not easy, nor does it happen independently. I would like to formally recognize the leadership teams of all our meetings for their diligence, commitment to excellence, and ongoing dedication to our educational programs. Education is the lifeblood of our Society. As we transition back to "live" meetings, let us not forget what we learned during the pandemic about how to collaborate virtually and when we come together.

At last year's Annual Meeting, I mentioned International Collaboration, Perioperative Medicine, and Leadership Development as three priorities for my Presidency. I want to review our progress in each of these areas.

During the ICCVA meeting in November 2021 (held virtually but nominally in Rome, Italy), we had a meeting at which Presidents of multiple professional Cardiac and Thoracic Anesthesia Societies from around the world came together to announce the foundation of a new organization for Cardiac Anesthesiology – the International Academy of Cardiac Anesthesiology (IACA). Conceptually this may be considered a "Society of Societies" as the only way an individual will be able to join will be via their local organization – be it a specialist society or where there is no such Society, an interest group of another more general Society.

Many Societies (ours included) have no geographical restrictions on membership, and this would also be a way to benefit from membership in IACA. This new

Continued...

PRESIDENT'S MESSAGE

"Leadership Development is a subject I'm deeply passionate about."

organization will bring our community closer and leverage the communication skills we all learned during the COVID 19 pandemic to achieve its goals. Virtual meetings, shared webinars, and online educational programming will be the initial tangible member benefits. We now move to address the logistics of forming the actual entity, organizing a governance structure, raising money, setting up administrative infrastructure, etc. Thank Dr. Alex Mittnacht, chair of our International Committee, for his vision, application, and ongoing commitment to IACA. Together with Dr. Fabio Guarracino, he has been instrumental in seeing this dream come to reality.

In terms of Perioperative Medicine, the Society continues to support the educational needs of our members who work outside the operating room in their professional commitments. We are engaging our surgical colleagues at AATS and STS to develop new ways our organizations can work together to promote excellence in Cardiac and Thoracic Perioperative Medicine. We are exploring the possibility of shared meetings with an emphasis on Critical Care and Perioperative Medicine and also whether it might be possible to hold our major events in the same city at the same time. The potential for cross-pollination of content and programming is self-evident. Co-location would allow teams to develop simulation programs to allow entire surgical and anesthetic teams from the same institution to train together. Look out for updates in this space when we firm up these exciting plans. Lastly, this year we are holding our inaugural Clinical Outcomes Research in Perioperative Medicine (COR-PM) symposium immediately before the Annual Meeting. Dr. Karsten Bartels has assembled an incredible faculty to teach this course and even wrote (and was awarded) an NIH grant to fund part of it. Congratulations to him and his team, and please be sure to join me in registering for and attending this great event.

My third priority is a subject I am deeply passionate about. Leadership development is a subject that, all too often, folks only begin to think about after they are appointed to their first leadership position. We need to address this, and SCA is committed to providing leadership training for our members, our committee members and Chairs, our Board of Directors, and course, our residents and fellows. We mandate unconscious bias training for all our leadership team and post online material at SCA University to permit all our members to access this critical content. This year, at our Board of Directors' meetings, we have held workshops on Communication Styles and Emotional Intelligence. I remain 100% committed to increasing the opportunities available to all our people to help them grow this essential and highly learnable skill. Technical proficiency does not automatically bring leadership proficiency, and as Marshall Goldsmith has famously said, "What got you here won't get you there." We are all leaders who can learn every day, and I believe that this investment in our Society's members and leaders will pay rich dividends in the future.

I will close this message with a couple of announcements for Presidential Task Forces for the 2022/23 season. I have asked Dr. Miklos Kertai and Dr. Linda Shore-Lesserson to lead a task force charged with developing a "how to do it" manual to provide a playbook for developing and writing documents collectively known as "clinical advisories." Such document types include clinical guidelines, practice advisories, clinical updates, systematic reviews, etc. There is a clear need for some high-quality instructional material about doing this effectively. Our members rely heavily on these documents for shaping their daily clinical practice, which is why

PRESIDENT'S MESSAGE

it is vital they are prepared thoroughly, in line with best practice, and based on the best sources of information available. I am 100% certain this task force will deliver us a product that will be not only a single source of truth for these documents but also one that will be very useful for members as they review the literature in the future.

Dr. Glenn Gravlee will lead the second task force I am announcing in this message. I have asked him to assemble a group of colleagues to collect, curate, organize, and store the "History of the SCA." We are 45 years old next year, and we must document our history and collect any memorabilia that members may have for those who come after us to appreciate. Glenn has served our Society in every role we have, and he has graciously agreed to lead this team. We will report back at our Annual Meeting in 2023, and in the meantime – anyone with any information to share about our history shouldn't hesitate to get in touch with Glenn through the Society's admin team.

I hope to see as many of you as possible in Palm Springs and thank you very much for the opportunity to serve as President of this fantastic Society.

Sincerely,

Andrew Shaw

Karsten Bartels MD, MBA, PhD
Chair, COR-PM 2022 Scientific Program

Anthony Bonavia MD

Kimberly Howard-Quijano MD, MS, FASE

Nadia Lunardi MD, PhD

Katie Schenning MD, MPH

Shahzad Shaefi MD, MPH

Jochen Steppan MD

Eric Sun MD, PhD

Brittney Williams MD

Meghan Prin MD

COR-PM 2022

Join Us for the First COR-PM Conference

Dear Colleagues,

The Society of Cardiovascular Anesthesiologists is thrilled to announce the first-ever Cardiovascular Outcomes Research in Perioperative Medicine (COR-PM) conference.

COR-PM is a completely new conference. The program was drafted by a group of diverse early- and mid-career anesthesiologists.

- **Advance** your understanding of high-quality clinical outcomes research within the T2-T4 translational spectrum.
- **Provide** mentorship capacity for early- and mid-career participants by providing a small-sized conference that permits "face time" with recognized leaders in the field, including Drs. PJ Devereaux, Dan Sessler, Jessica Spence, Monica Vavilala, Eric Sun, and many more.
- **Create** a personal, inclusive, and welcoming environment.

We look forward to seeing everyone in Palm Springs!

Alessia Pedoto MD
Chair, TAS Planning Committee

Randal S. Blank MD PhD
Vice Chair, Abstract Coordinator

Rebecca Y. Klinger MD MS
Workshop & PBLD Coordinator

TAS 2022

We're Back Live — Don't Miss Out!

Message from the TAS Program Committee

Dear Colleagues,

The TAS Planning Committee is looking forward to seeing you in sunny Palm Springs, CA, for the 10th Annual Thoracic Anesthesia Symposium.

May 13, 2022, marks an important date in the history of the Thoracic Anesthesia Symposium, and we are thrilled to share this milestone with all of you. During the past two years, the COVID pandemic and its aftermaths have posed challenges both at work and at home, forcing us to adapt and create a new "normal." The virtual format has taken over our practice and lifestyle because we may have forgotten what traveling and in person activities look like

May 13, 2022, will mark the start of a new "old routine"; we are planning to see each other in person for a time of learning and networking, seeing old friends and making new ones, and celebrating the 10th anniversary of TAS.

Please join us in sunny California for a day of lectures, workshops, and mentoring through both PBLDs and resident/fellow sessions. Join us for a town hall discussion on anything you may want to explore or share with our panel of experts or with your colleagues.

Be sure to support and encourage our fellows and residents at the abstract/poster stations and during the "best case" and "best research" sessions.

We hope to see you in person, full of energy and enthusiasm for a great in-person event. Come, participate, evaluate, and give us your candid feedback. We are excited to offer you updates, controversies, and new practices in the field of thoracic anesthesia. You are the foundation for the success of this day. Without you, we could not reach this 10th anniversary, but we will continue to grow and advance the field with you.

Thanks to your ongoing interest, participation, constructive feedback, and passion for thoracic anesthesia, the Thoracic Anesthesia Symposium has grown to be ten years old!

We are looking forward to meeting you all in California.

TAS Planning Committee

Kathy E. Glas, MD MBA FASE CME, *Committee Chair*
Gianluca Paternoster *EACTAIC Liaison*
Hyun Joo Ahn, MD PhD
Diana Anca, MD
Archer Martin, MD
Ju-Mei Ng, MD
Massimiliano Meineri, MD
Wanda M. Popescu, MD
Emily Teeter, MD FASE

Join us for these in-person workshops

TAS Meeting Highlights

Workshops Offered:

- Lung Isolation
- Thoracic Ultrasound Diagnosis
- Regional Anesthesia
- Critical Procedural Skills

Problem Based Learning Discussions (PBLDs) Offered:

- Lung Transplantation Management
- ECMO In Thoracic Surgery
- How to Design and Implement a Thoracic ERATS Program at Your Hospital
- Patient on LVAD for Thoracic Surgery
- Airway Crisis in the Thoracic Surgical Patient
- Oxygenation in One-Lung Ventilation

Register for this one- day event to maximize your interaction between attendees and faculty!

Click Here to view the TAS agenda

The Thoracic Anesthesia Symposium Planning Committee is enthusiastically inviting the world of non-cardiac anesthesiologists to join us for an excellent opportunity to learn what is new in the profession!

TAS 2022

Sasha K. Shillcutt MD MS FASE
Chair, Scientific Committee 2022

Mary Beth Brady MD FASE
Vice Chair, Scientific Program 2022

Jonathan Ho MD FASE
Workshop & PBLD Coordinator Scientific Committee 2022

SCA 2022

We're Back Live and In-Person! Join us on May 14-17

Message from the Scientific Program Committee

Dear Colleagues,

The Scientific Program Committee is so excited to gather in person at the SCA 44th Annual Meeting and Workshops in beautiful Palm Springs, California. After a challenging year in medicine, we look forward to coming together and networking with you.

The SCA Annual Meeting and Workshops will update you on the latest cardiothoracic anesthesia information through fantastic plenary sessions, controversial panel discussions, pro-con debates, hands-on workshops, mentoring sessions, and problem-based learning sessions.

Come and learn from abstract presentations, the always popular Super Echo Panel and legendary Echo Jeopardy, and a special session from the experts on the new Cardiothoracic Anesthesiology Certification exam.

Plan to hear on hot topics such as updates in coagulation, what's new in mechanical support, and professional development topics such as leadership and mentorship.

While we were glad to see so many of you virtually in 2021, we are thrilled to welcome you to Palm Springs in May!

Scientific Planning Committee

Kathy E. Glas, MD MBA FASE
CME Committee Chair

Jacques (Prince) Neekankavil, MD
Director, Fellow & Resident Program

Tara R. Brakke, MD FASE
Coordinator, Fellow & Resident Program

Alessia Pedoto, MD
TAS Chair (Liaison)

Megan Chacon, MD
PoCUS Co-Chair (Liaison)

James (Jake) H. Abernathy MD, MPH
STS Liaison

Nadia Hensley, MD
QSL Liaison

Jochen (Danny) Muehlschlegel MD MMSC FAHA
Research Committee Chair (Liaison)

Shahzad Shaefi, MD
SOCCA Liaison

Jennifer Hargrave, DO
Online Education Sub-Committee Chair (Liaison)

Gianluca Paternoster, MD PhD
EACTAIC Liaison

Sharon McCartney, MD, FASE
Echo Week Liaison

Karsten Bartels, MD

Adam Dalia, MD MBA

Stephanie Ibekwe, MD

Candice R. Montzingo, MD FASE

Adriaan Van Rensburg, MD MCChB MMED FCASA FRCPC

Michele Sumler, MD FASE

Kelly Ural, MD

Michael Essandoh, MD FASE

Rhagavendra Govinda, MD MBBS

Jiapeng Huang, MD PhD

Jenny Kwak, MD FASE

Emily Methangkool, MD FASE

Mihai V. Podgoreanu, MD FASE

SCA 2022

Annual Meeting Highlights

SCA Annual Meeting and Workshops is only a few weeks away! The Scientific Planning Committee has been working hard and diligently to bring you one of the best live and in-person meetings for the SCA membership and cardiovascular field. See you there!

Workshop 1

PoCUS

Description: Practical point of care ultrasonography i s becoming more accessible to a growing number of providers. This expert-led workshop is centered on the basics of transthoracic echocardiography (TTE). In addition to cardiac evaluation, the workshop will cover the use of lung ultrasound, vascular access, shock states, as well as the FAST (Focused Assessment with Sonography in Trauma) protocol. Special emphasis is placed on clinical applications of these techniques, as well as tips and tricks for image acquisition of these various modalities of POCUS.

Workshop 2

Professional Development

Description: Ready for advancement but not sure of your next step? This unique, interactive workshop will integrate expertise from both the academic and business world to help SCA members navigate and succeed in both the academic and private practice landscape, with the goal of fostering future leaders. Specifically, attendees will work on skill development in networking, mentorship, negotiation, and presentation. Take homes include how to perfect the "elevator pitch", cultivate healthy mentor and sponsor relationships, negotiate for time and compensation, and create and deliver an effective presentation.

Workshop 3

Perioperative Pacemaker and Implantable Cardioverter Defibrillator Management

Description: This workshop will address the design, functions, and programming of implantable pacemakers and ICDs. We will also discuss the current guidelines for the perioperative management of CIEDs during non-cardiac surgeries. Using case-based discussions, in a small group format, participants will apply these concepts in perioperative device management including lead extraction procedures. This will include interactive hands-on use of device programmers to interrogate and perform basic programming of pacemakers and ICDs.

Workshop 4

Regional

Description: Get ready for the regional anesthesia workshop on chest wall blocks. In this hand-on workshop, participants will be introduced to the most up to date regional anesthesia techniques in cardiothoracic surgery. This expert-led workshop centers on the basics of the chest wall innervation, regional anesthesia techniques for different surgical approaches, advanced strategies for ultrasound image optimization, and understanding the best practices for patient safety. In this workshop, participants will get a first-hand experience in imaging the newest regional anesthesia techniques, tips on how to improve ultrasound guidance of these fascial plane blocks and they will be given the tools to incorporate regional anesthesia into their future practice.

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SCA ANNUAL MEETING

Workshop 5

Interventional

Description: Structural heart interventions are here to stay! Learn the intricacies of these percutaneous procedures: TAVR, edge-to-edge MV repair, and LAA exclusion. Leaders from the field will provide tricks of the trade and tips for troubleshooting. Special emphasis is placed on procedural steps, communication, TEE image correlation, and collaborative practice.

Workshop 6

Advanced TEE

Description: Do not let numbers scare you! Learn advanced quantification with the echo experts at this cased based, TEE workshop that will discuss advanced methods of quantitative cardiac assessment. Participants will learn to recognize the role of quantitative echocardiography in clinical decision-making and discuss real-world applications of quantitative analysis. Learn today and put these techniques into practice tomorrow.

Workshop 7

3D TEE

Description: Applications of 3D echocardiography have advanced greatly in the last few years. This technology offers unique and critical solutions to clinical problems. This virtual workshop provides practical, problem based, and easy to understand sessions to help physicians master necessary 3D skills for daily practice. 3D ventricular function quantification, detailed 3D valvular analysis and 3D procedural guidance will be reviewed with renowned echocardiography experts in the field.

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Earl Wynands Lecturer

(Un)Professional Behavior in the Cardiac Theatre: Surely Not Problematic in 2022?

Presenter:
Eric Jacobsohn MBChB, MHPE, FRCPC

Keynote Speaker

Developing Physician Leaders

Presenter:
Brian Bolwell, MD, FACP

Join Us for These Events:

BOM & EARLY CAREER INVESTIGATOR

Moderators: MaryBeth Brady, MD, FASE
Jochen Muehlschlegel, MD, MMSC, FAHA
Karsten Bartels, MD, PhD

ADULT CARDIAC ANESTHESIA CERTIFICATION

Moderators: Mary Beth Brady, MD, FASE
Andrew Shaw, MB, FRCA, FFICM

SUPER ECHO

Moderators: Candice Montzingo, MD, FASE
Stephanie Ibekwe, MD, MS

- **American Society of Anesthesiologists (ASA) Update**
- **Business Meeting and Awards**

Click Here to register and view the meeting agenda.

SCA 2022

Please support our industry partners by attending the coffee breaks and lunch with the exhibitors in the exhibit hall.

Thank You to Our Industry Partners

PLATINUM

Edwards

SILVER

PHILIPS

BRONZE

Chiesi

Medtronic

National Board of Echocardiography

HemoSonics
A Stago Group Company

octapharma

EXHIBITOR

acacia pharma

Anesthesiology News

DeRoyal

Envision Physician Services

GE

HAEMONETICS

La Jolla Pharmaceutical

MERCK

NAPA North American Partners in Anesthesia

PACIRA BioSciences, Inc.

RETIA Medical

SafePath Medical

TEAMHealth

TopMedTalk

Trevena

werfen

[Click Here](#) to view the agenda for dates and times.

SCA 2022

ECHO BOARD REVIEW

See you June 18th and 19th

Join us for the Echo Board Review

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

The Echo Board Review Course is scheduled for the following days:

Saturday, June 18
10:00am – 5:00pm CST

Sunday, June 19
10:00am – 5:00pm CST

More information forth coming. Please watch your email.

ECHO BOARD

2022 SCA Election Results

SCA is pleased to announce the following individuals who have been elected to Society leadership positions:

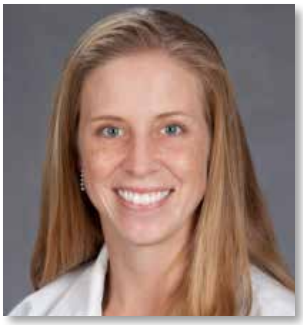
Director at Large

Daryl Oakes, MD
Stanford University

Director at Large (Re-elected)

Annemarie Thompson, MD
Duke University

Early Career Board of Director (Re-elected)



Jessica Brodt, MBBS, FASA
Stanford University

Early Career Board of Director

Stephanie Ibekwe, MD, MPH
Baylor College of Medicine

Continuing Medical Education (CME) Committee Member

Jenny Kwak, MD, FASA, FASE
Loyola University Medical Center

SCA's Outgoing Leaders — THANK YOU for Your Service

SCA would like to recognize the leaders whose terms of office have concluded. We greatly appreciate all their hard work towards improving our society, and we thank them for their involvement.

Board of Director 2021-2022

Michael P. Eaton, MD
University of Rochester

Early Career Board of Director 2019-2022

Emily Methangkool, MD, MPH
UCLA

CME Committee 2018-2022

Jennifer Hargrave, DO
Cleveland Clinic

Founding Officer Successor

Glenn Gravlee, MD
University of Colorado

Board of Director

Sasha K. Shillcutt, MD, MS, FASE
University of Nebraska Medical Center

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SF Match Fellowship Agreements Close June 2, 2022

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

The schedule for the 2023 training year is as follows:

Applicant Registration Began	Central Application Service Target/Deadline date	Rank List Submission and SCA Exception Agreement Deadline	Results Sent to Programs /Applicants and Medical Schools	Post-match Vacancies Posted	Training Position Starts
November 8, 2021	March 2, 2022	June 2, 2022	June 9, 2022	June 10, 2022	July, 2023

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

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

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

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

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

Program directors complete the first part of the match exception process. **Program directors — click here to begin.** You will need to log in with your SCA username and password. Once the program director completes this portion of the process, the applicant will receive an email with a link to the form they must complete.

Any match irregularities will be referred to the ACTA Fellowship Program Directors Council of SCA.

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2022 Kaplan Leadership Development Grant Recipients

Sheela Pai Cole, MD, FASE, FASA
Stanford University

Stephanie Ibekwe, MD, MPH, MS
Baylor College of Medicine

The Kaplan Leadership Development Award is sponsored by the SCA Endowment, and its mission is to assist cardiothoracic and vascular anesthesiologists in their careers by granting funding to further their focus on leadership opportunities and development through course work and leadership specific studies.

The Endowment covers $5,000, with the applicant's institution matching the funds, providing for a $10,000 grant. The Society of Cardiovascular Anesthesiologists is pleased to announce the 2022 recipients.

Save the Date

Rocky Mountain Valve Symposium July 21-22, 2022

Click Here for more information on the symposium.

Click Here to register for the event.

SOCIETY OF CARDIOVASCULAR ANESTHESIOLOGISTS
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MEMBER CORNER

AWEsome Woman Interview

Andra E. Duncan, MD, MS, FASE

Cleveland Clinic

Brief introduction about yourself:
I am an Associate Professor and Vice Chair of Research in the Department of Cardiothoracic Anesthesiology at the Cleveland Clinic where I have practiced for the past 21 years. I have enjoyed the busy clinical work at the Cleveland Clinic and the never-ending supply of complex cardiac surgical procedures in a high acuity patient population. This setting is also ideal to educate our talented trainees and mentor junior anesthesiologists in research, a privilege that I have enjoyed throughout my career.

1. What led you to become a Cardiovascular/Thoracic Anesthesiologist?
I was drawn to this career because I have always been fascinated with cardiovascular physiology. I enjoy the challenge of managing critically ill cardiac surgical patients through complex and difficult cardiac surgeries.

2. How did you hear about the SCA?
Hmmm. . . good question! I have been a member of the SCA for many years. I presented my first research abstract at a SCA meeting over 20 years ago. I must have heard about the SCA from my attending physicians during residency.

3. What roles have you held for the society?
I served on the SCA Scientific Planning Committee for 2 terms (4 years) and I am currently on my 3rd term on the SCA Research Committee (going on 6 years). I have moderated many sessions, participated in workshops, and gave several presentations at the podium at annual SCA meetings.

4. What is one of your greatest achievements as a cardiovascular/thoracic anesthesiologist?
My greatest achievement is mentoring residents, fellows, and junior staff in research projects that advanced the field of cardiothoracic anesthesia. I enjoy contributing to the professional development and growth of our future leaders.

5. Do you have any advice for fellows and residents?
Choose your career goals wisely. Then make a 1- and 5-year plan to reach them.

6. Have you experienced any difficulties as a woman in the field?
Being a "mom" and a busy cardiothoracic anesthesiologist was stressful: figuring out how to get the kids to baseball practice, making sure homework was done, and putting dinner on the table – all while working in a busy cardiac anesthesia practice was too much at times. Women often carry an extra load when they manage home and family responsibilities on top of career aspirations.

7. Do you have any advice for other women in the field?
You CAN have it all.

8. How do you balance work and personal life?
I still work long hours, but my kids are now in college. So, I finally have some "me" time. I try to relax when I get home from work. I am also working on my golf game.

9. What is something you enjoy doing outside of work?
Reading books, taking a walk, playing golf (poorly), travelling, spending time with family and friends.

10. Would you change anything about the path you took to get to where you are now?
If I could do it again, I would not hold back because I was a junior staff. I admire how some of the most junior colleagues have led big changes that impact our field.

11. What was the best piece of advice you received?
Your risk of rejection is 100% for papers/grants/proposals that you do not submit.

Surgical Approaches to Minimally Invasive Valve Surgery: A Guide for Regional Cardiac Anesthesiologists

Michael McGrath, BS and Peter Neuburger, MD, FASE
NYU Grossman School of Medicine, New York, NY

Commentary by
American Society of Regional Anesthesia (ASRA) RACER member, Richard K. Kim, MD MSc, Clinical Assistant Professor, Stanford University School of Medicine.

Regional and neuraxial anesthesia for cardiac surgery has evolved from a niche practice to one that has gained popularity beyond select medical centers in the United States. While spinal analgesia, epidural analgesia and the thoracic paravertebral (TPV) block are not new techniques, the recent development of novel fascial plane blocks including pectoralis (PECS) I and II blocks (2011, 2012), serratus anterior plane (SAP) block (2013), pecto-intercostal fascial (PIF) block (2014) and erector spinae plane (ESP) block (2016) largely contributed to widened acceptance.[1-5] In 2017, the Enhanced Recovery After Surgery (ERAS) Society first gathered a group of expert cardiac anesthesiologists and surgeons to formalize consensus recommendations which included the use of regional techniques.[6] After years of teaching these methods at the Annual Meeting & Workshops, in 2019 the Society of Cardiovascular Anesthesiologists further recognized this movement by forming the Regional Anesthesia for Cardiothoracic Enhanced Recovery (RACER) special interest group (SIG) to serves as a forum for member discussion.

The vast and growing number of regional techniques can be overwhelming. While there are numerous high quality review articles that cover the blocks used in cardiac surgery, nomenclature can be confusing and recently there has been an effort to standardize terms.[7,8] There is less discussion amongst cardiac anesthesiologists regarding the variations in surgical incision sites used in the most common types of minimally invasive cardiac surgery. While spinal, epidural, TPV and ESP blocks can provide broad analgesia for the thorax or hemithorax, a truly minimally invasive anesthetic approach aims to provide only necessary sensory coverage, while minimizing unnecessary physiologic effects. An understanding of surgical anatomy ultimately helps the anesthesiologist select a proper regional approach. On behalf of RACER SIG, this article will discuss the location and anatomy of surgical incisions commonly used in minimally invasive valve surgery involving the lateral chest wall.

Surgical Aortic Valve Replacement via Right Anterior Mini-Thoracotomy

The first surgical aortic valve replacement (SAVR) via a right anterior mini-thoracotomy (RAT) was performed in 1993.[9] Along with the upper hemisternotomy, this remains a common minimally invasive approach.[10] This technique begins with a 4-6 cm skin incision in the 2nd or 3rd right intercostal space (ICS) near the sternal border, after which soft tissue retractors and rigid retractors with narrow blades provide visualization of the operative site (Figure 1). Following pericardiotomy, cannulation for cardiopulmonary bypass (CPB) is established peripherally by percutaneous or cut-down access of the common femoral vessels or centrally via direct cannulation of the ascending aorta and right atrium.[11] A low-profile aortic cross-clamp is placed either via the primary incision or through a separate stab incision inferolateral to the right clavicle (2).[12] Ascending aortic endoballoon occlusion can also be used via a femoral arterial cannula. Following completion of the procedure, a small chest drainage tube is placed in the right pleural space through a separate intercostal space.

Given the relatively medial incision site of SAVR via RAT, a regional anesthetic must be chosen carefully. The superficial parasternal intercostal plan (PIP) block, a standardized term

used to encompass the PIF block, transversus thoracis plane block, parasternal pectoral block and others, would be appropriate.[8] This block targets the anterior cutaneous branches of the intercostal nerve responsible for innervation from the sternum to the midaxillary line. Case reports have demonstrated successful use of the superficial PIP block following RAT SAVR to provide analgesia from the sternum to the midaxillary line, effectively reducing postoperative opioid administration and postoperative numerical pain scores.[13]

Minimally Invasive Mitral Valve Repair and Replacement Surgery

Minimally invasive lateral approaches have been similarly embraced in the realm of mitral valve repair (MVr). The most common approach is a right mini-thoracotomy under direct visualization. The primary thoracotomy incision is 5-8 cm and placed in the 4th right ICS, inferolateral to the areola in men and in the submammary crease in women.[14] A soft tissue retractor with or without a small thoracic retractor is used to then spread the ribs. Optionally, a thorascope is introduced through the 2nd right ICS. The aorta can be cross clamped via a transaxillary approach in the 2nd/3rd right ICS using a Chitwood clamp, through the primary incision with a flexible clamp or via the femoral artery with ascending aortic endoballoon occlusion. Finally, a left atrial retractor is inserted parasternally to expose the valve. Like SAVR via RAT, some centers prefer peripheral femoral cannulation for MVr, which may allow for better exposure, while others utilize direct aortic cannulation to avoid the risks of retrograde arterial perfusion.

Robotic MVr presents the least invasive technique for surgical treatment of mitral valve disease, with the largest incision as small as 12-20 mm (Figure 2).[15] Generally, the working (access) port is created in the 4th right ICS anterior axillary line. The left arm port is placed 2 interspaces cranial while the right arm port is placed 2 interspaces caudal, both on the anterior axillary line. Finally, the camera port is placed anteriorly in the same ICS as the working port while the LA retractor port is placed in the 4th or 5th ICS, medial to the midclavicular line.

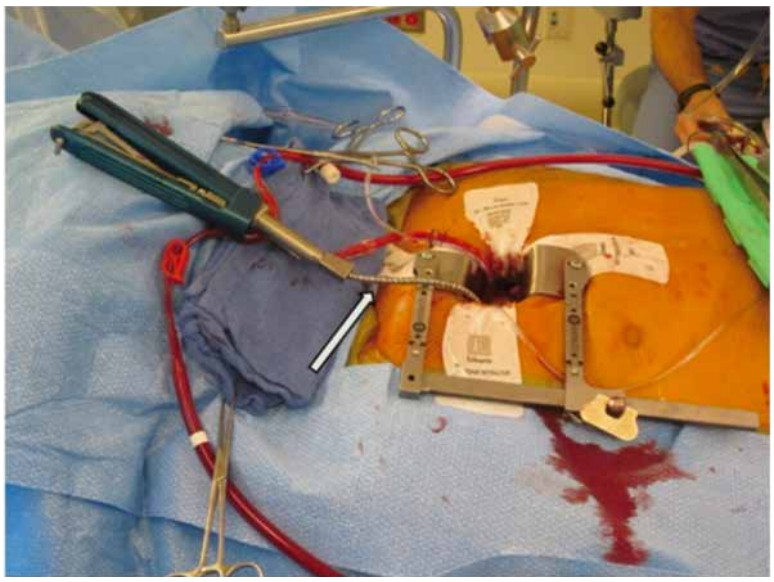
Because the primary incision is lateral in both of these MVr approaches, the optimal regional technique is different than for RAT SAVR. The superficial or deep SAP blocks is may be appropriate, as they provide analgesia from T2-T7 with variable spread to T9.[3] This approach spares the anterior cutaneous branches of the intercostal nerves and therefore has limited coverage medial the midclavicular line. The pectoserratus plane block, previously referred to as PECS II, may also be considered The principal targets of this block are the medial pectoral, lateral pectoral, long thoracic, thoracodorsal nerves, with possible medial effects via the anterior branches of intercostal nerves. This technique provides excellent coverage of the axilla and T1-T4 dermatomes with variable spread to T6.[16] Depending on placement of accessory ports, these blocks may require supplementation with a superficial PIP block, or alternatively a TPV or ESP block can be used. A recent review specifically highlighted the use of SAP or pectoserratus plane blocks in the setting of robotic MVr, though the authors stated that pre-induction TPV block was their analgesic preference.[17] Multi-level TPV Block has demonstrated efficacy in reducing intraoperative and postoperative opioid requirements as well as postoperative pain scores following robotic MVr when compared to control.[18]

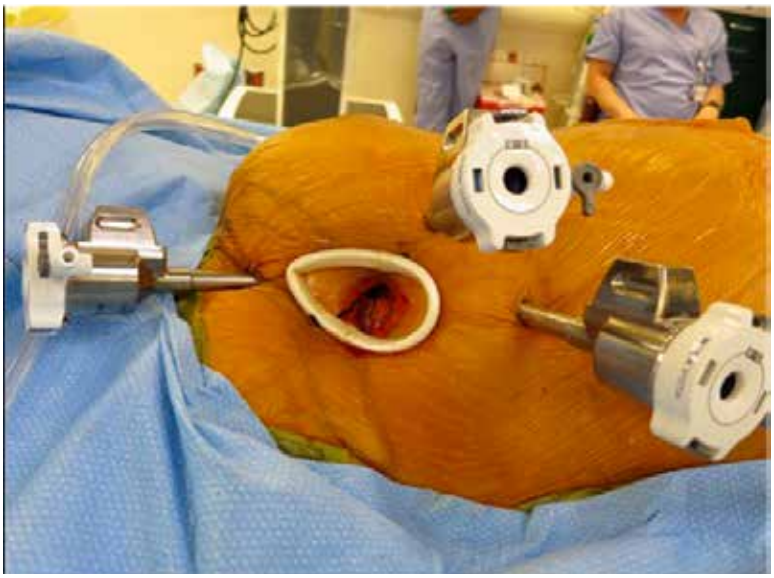
Transapical Approaches to Transcatheter Valve Procedures

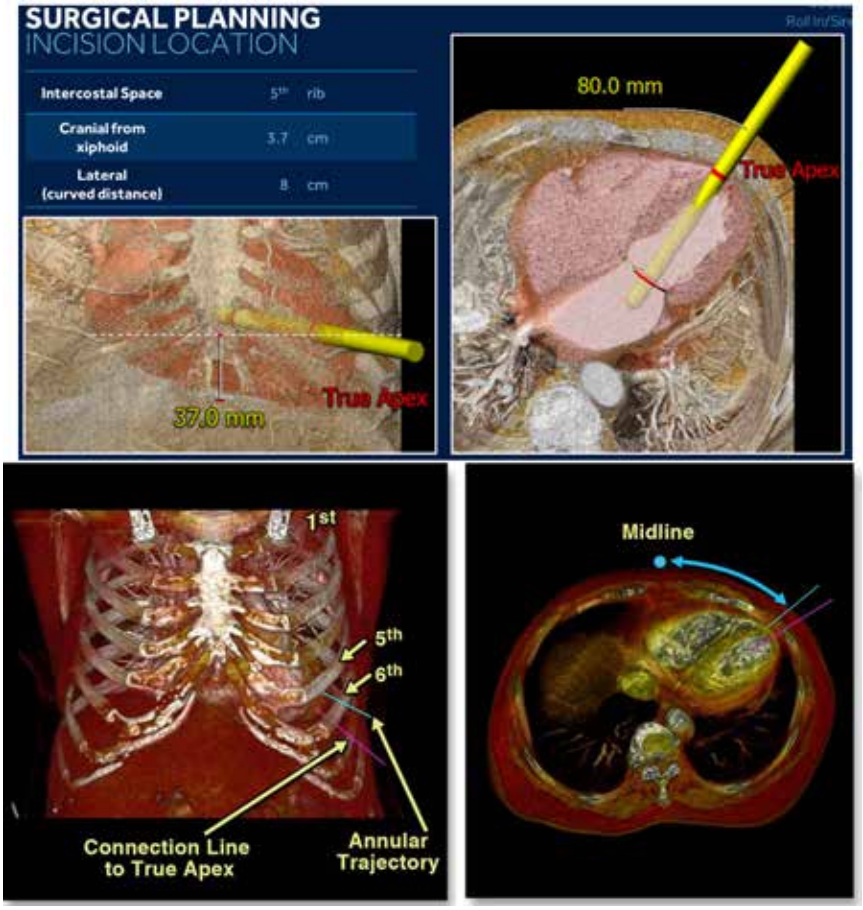
Transapical (TA) transcatheter aortic valve implantation (TAVI) was a frequently used approach in patients with unfavorable transfemoral access anatomy. Although infrequently used in contemporary TAVI due largely to a reduction in sheath size, the TA approach has been used more recently in transcatheter mitral valve interventions, including chordal repair with the NeoChord DS1000 system or valve replacement with the SAPIEN 3 and Tendyne systems. The TA approach uses a left anterolateral mini-thoracotomy to gain direct anterograde access to the left ventricle. Identification of the optimal interspace is frequently guided by preoperative computed tomography, or with surgical palpation and transthoracic echocardiography (Figure 3). A 5-7 cm skin incision is usually made in the 5th or 6th left anterolateral ICS on the anterior axillary line.[19] The use of soft tissue retractors and rigid retractors allows apical access which permits transapical sheath insertion and valve intervention under echocardiographic and fluoroscopic guidance.

The left mini-thoracotomy for a transapical approach lends itself to most of the blocks previously described for MVr. TPV block has been shown to reduce opioid administration and decrease incidence of atrial fibrillation after TA TAVI with limited impact on hemodynamic stability.[20, 21] SAP or pectoserratus plane blocks have also been used in case studies.[22, 23]

Traditional cardiac anesthesia utilized systemic opioids can treat postsurgical pain regardless of the surgical approach, albeit with unnecessary side effects. Likewise, neuraxial anesthesia can provide near complete coverage of somatic pain receptors, at the expense of profound vasodilation and increased procedural risk. The modern ERAS cardiac surgery approach calls for a "less is more" technique, and only through a complete understanding of surgical anatomy can the most appropriate regional block be selected by the cardiac anesthesiologist.







Commentary by

American Society of Regional Anesthesia (ASRA) RACER member, Richard K. Kim, MD MSc, Clinical Assistant Professor, Stanford University School of Medicine.

Since the inception of RACER SIG in ASRA and SCA in 2019, there has been an explosion of interest in matching the analgesic needs of minimally invasive cardiac surgery with regional anesthetic techniques beyond the neuraxis, as wonderfully narrated above. Mr. McGrath and Dr. Neuburger provide an excellent timeline of recently described fascial plane blocks, with careful descriptions of minimally invasive surgical approaches with which these blocks may marry well. In addition to the anterior mini-thoracotomy, the authors point out common locations of accessory ports, which can be additional sources of nociception. Indeed, the block needle's reach has literally come full circle along the chest wall (Chin et al.). As evidence continues to build on these techniques' effects on pain scores and opioid consumption, how can we simultaneously demonstrate their effectiveness and value? Waiting for randomized, controlled, multicenter trial data must be weighed with reports of successful integration of chest wall blocks into enhanced recovery after cardiac surgery (ERACS) protocols from hospitals around the world (Sondekoppam et al.).

A team-based approach in a culture of collaboration and communication is crucial for any enhanced recovery pathway, no less for the incorporation of chest wall blocks into cardiac surgery (Kim et al.). Engaging the surgeon early as a stakeholder can allow cardiac and regional anesthesiologists for any block-related concerns to be addressed and for shared agreements on specific, targetable outcome goals, whether that be facilitating fast-track extubation, decreasing opioid consumption, and/or accelerating transfer from the intensive care unit to the ward. Furthermore, deciding on the block based on any discussed variation from the aforementioned surgical approaches can encourage further

buy-in. If a dedicated regional anesthesia service is available, the cardiac and regional anesthesiologists can decide on the choice and technique of block placement. For example, a single local anesthetic injection versus a continuous infusion catheter may be considered. A separate block team can further optimize the timing of the block, especially if decreases in intraoperative opioid administration and fast-track extubation are also desired.

Follow-up can further provide quality assurance while enhancing the visibility and value of a perioperative anesthesia service. Additional stakeholders in the intensive care unit and the surgical wards include intensivists, nursing staff, hospitalists, and additional allied healthcare professionals. Education on the regional analgesia techniques must happen, including clear expectations on the scope of analgesia, as well as issues that warrant further consultation from the regional anesthesia and acute pain medicine team (e.g., block disconnect, pump programming, pain not covered by block). This is particularly important for ERACS pathways that utilize regional analgesia catheters which facilitate continuous infusions or intermittent boluses, which merit daily management until catheter removal. As a watched kettle is oft-said to never boil, a well-managed nerve block catheter may improve outcomes (and never fail). Close tracking of the blocks' effects on outcomes are important to further demonstrate their cost-saving potentials. For instance, if a serratus anterior plane block can facilitate earlier extubation in the OR and decrease the staffing need for respiratory therapists postoperatively, is this not valuable and worth making the block work? (Mariano ER)

Minimally invasive cardiac surgery has created a wealth of opportunities for improving healthcare outcomes across the perioperative spectrum. As Mr. McGrath and Dr Neuberger point out, the extremes of neglecting postoperative pain in the cardiac surgical patient and risking unnecessary complications with neuraxial analgesia can be circumvented by "minimally invasive" regional anesthesia that maximizes results. Cardiac and regional anesthesiologists look forward to engaging with both the broader cardiac and regional anesthesia communities to share best practice insights and to advance clinical practice.

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The Effect of the Cystic Fibrosis Care Center on Outcomes after Lung Transplantation for Cystic Fibrosis

Bush E, Krishnan A, Chidi A, et al. *The Journal of Heart and Lung Transplantation*. 2022 Mar;41(3):300-307.

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Background

Although recent advances in Cystic Fibrosis (CF) therapies have prolonged life expectancy, these patients still possess a significant risk of developing end stage lung disease (ESLD). Transplantation for these patients should be considered when the two-year mortality is greater than 50% or patients demonstrate New York Heart Association class 3 or 4 symptoms.[1] CF is the leading cause of suppurative ESLD and is the underlying etiology for 15.4% of all lung transplants.[2] The Cystic Fibrosis Foundation (CFF) established an accredited care center network in 1961 with the focus on treating CF.[3] Cystic Fibrosis accredited care centers (CFCC) provide care to CF patients through dedicated specialists focused on multi-system disease management. Bush and colleagues recently investigated the outcomes of CF patients undergoing bilateral lung transplants at both CFCC and non-CFCC transplant centers.

Methods

The study aimed at comparing outcomes in CF patients who underwent lung transplant at a CFCC and with those patients who received transplant at a center without the CFCC designation. The authors conducted a review from 2005-2018 of all adult primary bilateral lung transplant recipients with CF listed in the Scientific Registry of Transplant Recipients. The primary outcome was graft failure however waitlist mortality, time to transplant, and comparison of outcomes with center volume were also reported. During the thirteen-year study period, 2,573 patients with CF underwent primary bilateral lung transplantion at 68 different lung transplant centers. Of the 68 centers, 50 centers were designated as CFCC and those centers transplanted 2,263 patients (87.9%). Notably, median annual transplant volume at CFCCs was pointedly higher (5.6 vs. 2.5 transplants/year, $p < 0.001$). Follow up for the study period was a median of 1,113 days.

Results

Adjusting for cofounders, there was a statistically significant decrease in the risk of graft failure ($p < 0.001$) for patients who were transplanted at a CFCC. Graft failure was the number one cause of death and African American race as well as mechanical ventilations prior to transplant increased the risk of graft. Mortality was 35.4% (911) during the study period and 5.9% (152) of the patients required re-transplantation. There were no reported significant differences between CFCCC and non-CFCC centers when measuring time to transplant and waitlist mortality. Survival for those patients transplanted at a CFCC was 7.8 years compared to 4.4 years for those patients transplanted at a center without CFCC designation ($p < 0.001$).

Discussion

The importance of multi-disciplinary coordination within lung transplantation has been described in the literature, both in general and specific to recipient ESLD.[4-6] Within CF, this team-based coordination is best found at one of the CFF-accredited centers. Not only was there a 33% risk reduction in mortality or severe morbidity when performed

at a CFCC, it was also independent of volume – highlighting the importance of a well-functioning team on overall outcomes.

The coordination of team care must be based on high-quality data and expert consensus, as has been seen recently in the International Society of Heart and Lung Transplantation (ISHLT)/SCA joint consensus on anaesthetic management[7], the ISHLT lung transplantation connective tissue disease guidelines[8], and the forthcoming ISHLT expert consensus on perioperative utilization of ECLS in lung transplantation. Future clinical and research efforts to optimize this team care in cardiothoracic transplantation anesthesiology should focus on developing a coordinated strategy that is tailored to recipient ESLD, built upon the foundational principles of attenuating primary graft dysfunction and technical complications, and multidisciplinary in composition.

The retrospective nature of this study as well as examination of a general database provide significant limitations in drawing absolute conclusions about the ideal care of a CF patient presenting for lung transplantation. However, this manuscript is of note because it represents a further step in the direction highlighting the positive influence of comprehensive perioperative care directed towards recipient ESLD on overall lung transplantation outcomes.

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LITERATURE REVIEWS

Individualized or Liberal Red Blood Cell Transfusion After Cardiac Surgery: A Randomized Controlled Trial

Fischer MO, Guinot PG, Debroczi S, et al. Individualized or liberal red blood cell transfusion after cardiac surgery: a randomized controlled trial. Br J Anaesth 2022; 128: 37-44.

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Background

Red blood cell (RBC) transfusions are frequently administered to cardiac surgery patients to maintain oxygen delivery to tissues. The determination of when to transfuse RBCs has predominantly been guided by hemoglobin level. RBC transfusions have risks include acute hemolytic reactions, transfusion associated circulatory overload (TACO), transfusion-related acute lung injury (TRALI) and delayed reactions as well. Therefore, a more restrictive, physiologic transfusion strategy has the potential for fewer adverse reactions.

Currently, trials have shown that a more restrictive transfusion strategy is not inferior to a more generalized, liberal strategy after cardiac surgery but there is no hemoglobin threshold that has been consistently recommended.[1,2] In addition, it is also important to consider the physiologic condition of the patient, specifically the oxygen delivery versus oxygen consumption.[3] Central venous oxygen consumption (ScvO2) can be calculated from the following equation: ScvO2 = SaO2 - (VO2/DO2). ScvO2 therefore considers oxygen delivery and oxygen demand rather than hemoglobin level alone. More specifically, after optimization of cardiac output and oxygenation, ScvO2 communicates individual tolerate to anemia. The study sought to investigate whether a more physiologic approach to RBC transfusion with the use ScvO2, rather than the traditional approach with an arbitrary threshold of hemoglobin level, particularly post-cardiac surgery, was feasible and justified.

Methods

This study involved an open label, randomized, controlled trial with two groups at two French academic hospitals. Both groups were patients greater than 18 years of age admitted to the intensive care unit after undergoing cardiac surgery with cardiopulmonary bypass. In addition, both groups had hemoglobin levels < 9 g/dL. Patients were randomized to receive either one unit of RBCs (hemoglobin group) or transfusion only if the ScvO2 was < 70% after correction for hypoxemia and hypovolemia as necessary (individualized group). The individualized group could also receive a transfusion if the hemoglobin decreased to less than 7.5 g/dL (with an ScvO2 > 70%) for safety reasons.

The primary outcome was the number of patients receiving a transfusion. Secondary composite outcomes included acute kidney injury, cerebrovascular accident, myocardial infarction, acute heart failure, mesenteric ischemia, or in-hospital mortality. At one- and six-months, mortality data were collected.

Results

The final study comprised 164 patients with 80 patients in the hemoglobin group and 77 patients in the individualized group (1 protocol violation and 6 participants withdrew). In comparing the hemoglobin and individualized groups, there were not any statistically significant differences including preoperative data, surgical risk, type of surgery and intraoperative characteristics. Similarly, there were no significant difference between preoperative hemoglobin concentrations (12.3 and 12.2 g/dL), hemoglobin concentrations at inclusion (8.2 and 8.2 g/dL), or baseline ScvO2 (63% and 62%) for the hemoglobin and individualized groups, respectfully.

The study investigators used an intention-to-treat analysis. In terms of the primary outcome of the study, fewer patients in the individualized group received a transfusion than in the hemoglobin group (79% versus 100%). There were no significant differences in the secondary outcome between the two groups and there was a non-significant difference in mortality at one and six months. Further, the investigators performed a sensitivity analysis to determine the number of patients receiving RBC transfusion if the hemoglobin threshold was 8 g/dL instead of 9 g/dL and found that 22 subjects would have received one unit of RBCs in the hemoglobin group and 15 subjects in the individualized group ($p<0.001$).

Discussion

A restrictive transfusion strategy has previously been shown to be non-inferior to a liberal transfusion strategy in terms of postoperative mortality.[1,2] However, there is much debate regarding what this hemoglobin threshold should be with international guidelines ranging in consensus anywhere from 7.0 g/dL to 10 g/dL.[4,5] The higher suggested thresholds may be explained by data indicating that certain patients, such as those with symptomatic coronary disease or using beta blockers, may require a more liberal transfusion strategy.[6]

Yet still, it is important to consider the physiologic condition of the patient, specifically the oxygen delivery versus oxygen consumption. When controlling for hypovolemia and hypoxemia, ScvO2 serves as an excellent metric for individual oxygen delivery and demand. Using this individualized strategy as a transfusion trigger, the authors found it to be non-inferior to the use of a hemoglobin threshold in post-cardiac surgery patients in terms of post-operative morbidity or mortality. Further they were able to demonstrate that this type of strategy reduced the number of patients receiving at least one RBC transfusion as compared to the strategy based solely on hemoglobin levels.

Limitations of this study include the higher hemoglobin threshold than recommended in more recent guidelines, which suggest 8 g/dL rather than 9 g/dL. In addition, the primary outcome of this study may have been more informative if it was mortality or other clinical outcomes rather than the number of RBC transfusions administered.

In summary, the authors propose the use of ScvO2 in transfusion strategies for post-cardiac surgery patients to decrease the number of RBC transfusions and transfusion adverse events while also providing a more individualized, physiologic transfusion guide.

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4-Year Outcomes After Left Atrial Appendage Closure Versus No Warfarin Oral Anticoagulation for Atrial Fibrillation

Osmancik P, Herman D, Neuzil P, Hala P, Taborsky M, Kala P, Poloczek M, Stasek J, Haman L, Branny M, Chovancik J, Cervinka P, Holy J, Kovarnik T, Zemanek D, Havranek S, Vancura V, Peichl P, Tousek P, Lekesova V, Jarkovsky J, Novackova M, Benesova K, Widimsky P, Reddy VY; PRAGUE-17 Trial Investigators. J Am Coll Cardiol. 2022 Jan 4;79(1):1-14.

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Background

Left-atrial appendage closure (LAAC) devices, including the Watchman or Amulet devices, have been used as a nonpharmacologic alternative to anticoagulation with warfarin or direct oral anticoagulants (DOAC) in patients with atrial fibrillation (AF) at high risk of stroke. These devices could theoretically provide similar reductions in stroke compared to anticoagulants with a lower risk of bleeding. There are few long-term trials, however, and the 2 available randomized controlled trials with long-term data compared the Watchman device to warfarin.[1]

Given the increased usage of DOACs, which have largely replaced warfarin in clinical practice, the PRAGUE-17 trial was prompted to compare LAAC devices to DOACs. This trial's primary analysis demonstrated LAAC devices were noninferior to DOACs in rates of stroke, systemic embolism, clinically significant bleeding, or cardiovascular death at a median follow-up of 19.9 months.[2] In their latest manuscript, "4-Year Outcomes After Left Atrial Appendage Closure Versus Nonwarfarin Oral Anticoagulation for Atrial Fibrillation," the same authors present the 4-year outcomes of the PRAGUE-17 trial recognizing that the maximum benefit in terms of bleeding would be expected to be even long-term.[3] Their aim, like the prior study, was to determine noninferiority of LAAC devices to DOACs in rates of stroke, systemic embolism, clinically significant bleeding, and cardiovascular death.

Methods

This study was an investigator-initiated, multicenter, prospective, open-label, randomized, noninferiority trial conducted at 10 cardiac centers in the Czech Republic. Patients with non-valvular AF and moderate or high risk for stroke or bleeding were eligible. This included patients with a history of bleeding requiring intervention or hospitalization, cardioembolism while on anticoagulation, and a moderate to high risk profile based on CHA2DS2VASC ≥3 plus HAS-BLED ≥2. They excluded patients with mechanical valves, mitral stenosis, comorbidities aside from atrial fibrillation that required anticoagulation, patent foramen ovales with a large atrial septal aneurysm, symptomatic carotid stenosis, significant bleeding within the last 30 days, cardioembolic event within the last 30 days, or a creatinine clearance less than 30 mL/min. In the LAAC device group, if the patients had a preexisting LAA clot on TEE at the time of device placement, they were also excluded.

The two treatment groups were (1) LAAC device + dual anti-platelet therapy (DAPT) with clopidogrel 75 mg and aspirin 100 mg per day for 3 months followed by aspirin indefinitely or (2) DOACs at the manufacturer-recommended dose. The devices used were the Amulet (Abbott Inc), Watchman (Boston Scientific Inc), or Watchman FLX (Boston Scientific Inc). The patients in the DOAC group received apixaban, rivaroxaban, or dabigatran indefinitely. Follow-up was at 6 weeks; 3, 6, 9, 12 months; and every 6 months thereafter.

The primary outcome was a composite of safety and efficacy characteristics of both strategies, including stroke, systemic embolism, clinically significant bleeding, cardiovascular (CV) death, and significant periprocedural or device-related bleeding. This primary endpoint was analyzed as a modified intention to treat. Secondary outcomes were clinically significant bleeding and non-procedural clinically relevant bleeding.

Results

After enrolling 415 patients, 13 patients were excluded, and 201 patients were ultimately randomized to each group. Baseline characteristics were similar between the LAAC device group and DOAC group. In the LAAC device group, 61.3% received an Amulet, 35.9% received a Watchman, and 2.8% received a Watchman-FLX. Thirty-three patients in the LAAC device group were ultimately placed on a DOAC during the trial. In the DOAC group, 95.5% of patients were placed on apixaban along with 4% and 0.5% being placed on dabigatran and rivaroxaban, respectively.

The composite primary outcome of stroke, systemic embolism, clinically significant bleeding, CV death, and significant periprocedural or device-related bleeding was not statistically significantly different between the two groups with the noninferiority P value = 0.006. The incidence of non-procedural clinically relevant bleeding was lower in the LAAC group, P = 0.038 (HR 0.55; 95% CI = 0.31-0.97). These results were consistent between the modified intention to treat analysis, post hoc per-protocol analysis, and post hoc on-treatment analysis.

Discussion

This study was a long-term analysis of the PRAGUE-17 trial, which was a noninferiority study comparing LAAC devices to DOACs at the 4-year timepoint. The primary composite outcome was similar among the two groups and consistent between the modified intention to treat analysis, post hoc per-protocol analysis, and post hoc on-treatment analysis. The LAAC device group had a lower incidence of non-procedural clinically relevant bleeding. This separation in bleeding events between these groups appeared to increase in favor of the LAAC device group as the study progressed. These data are consistent with the previous studies PROTECT-AF and PREVAIL, which compared LAAC devices to warfarin over a 5-year period. Both studies showed noninferiority in their primary composite outcomes as well as fewer bleeding events in the LAAC device groups (HR: 0.48; 95% CI 0.32-0.71).[1]

No trials thus far have studied percutaneous LAAC devices with oral anticoagulation (OAC) against OAC alone for superiority. The LAAOS III trial, however, showed superiority of surgical LAAC plus OAC against OAC alone.[4] A similar trial is reasonable to test LAAC devices combined with OAC against OAC alone in patients we a lower risk of bleeding to determine and additional benefit in the combined group.

This study was underpowered to detect differences between individual outcomes that comprised the composite outcomes (stroke, systemic embolism, clinically significant bleeding, CV death, and significant periprocedural or device-

related bleeding). The composite endpoint also included both bleeding and thromboembolism, which likely have competing directions of effect. Additionally, 12.9% of patients in the DOAC group discontinued treatment, which may have contributed to the noninferiority of the LAAC device group. The authors argue, however, that this may better reflect true practice as other studies have shown up to 21.7% of patients discontinued their OACs during trials.[5]

Conclusion

The 4-year outcomes for the PRAGUE-17 trial demonstrate continued noninferiority of LAAC devices to DOACs for the composite outcome of stroke, systemic embolism, clinically significant bleeding, CV death, and significant periprocedural or device-related bleeding. LAAC devices also demonstrated lower a lower incidence of non-procedural bleeding that widened as the study progressed.

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Anatomical and Technical Predictors of Three-Dimensional Mitral Valve Area Reduction After Transcatheter Edge-To-Edge Repair.

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Background

Based on the surgical Alfieri stitch technique, clinical use of mitral valve transcatheter edge-to-edge repair (TEER) for treatment of mitral regurgitation (MR) has expanded rapidly since the first human case in 2003. MitraClip (MC) was the first TEER device and remains the most widely used. TEER devices use a leaflet repair method that apposes the anterior and posterior leaflets at the site of the regurgitant jet, improving coaptation while creating a double- or triple – orifice, depending on the number of clips implanted. Society guidelines state TEER is a Class IIA recommendation in patients with primary severe MR and high surgical risk or greater, or with chronic severe secondary MR related to LV systolic dysfunction with persistent symptoms.[1] One of the principal limitations of TEER technique is the reduction of mitral valve area (MVA) which may theoretically result in post-procedural mitral stenosis (MS), defined as 3D MVA < 1.5 cm2.[2] Due to the concern for iatrogenic MS, pre-operative MVA ≤4 cm2 has been a relative contraindication to TEER.[3] However, the EVERST anatomical criteria is based on two dimensional MV assessment and little is known about the predictors of MVA reduction.[4] Using 3D transesophageal echocardiography (TEE) this study investigated functional, anatomical, and technical factors influencing MVA reduction after implantation of one or two mitral clips.[5]

Study Design

This study was a single center prospective observational study of patients with symptomatic MR undergoing treatment with TEER. The authors aimed to evaluate functional, anatomical, and technical factors influencing MVA reduction after the implantation of one or two mitral clips with the goal to derive the minimal MVA required to prevent the development of clinically significant MS after TEER. The study included 166 consecutive patients treated with NTR or XTR MitraClip (Abbott Vascular, Abbott Park, IL) between 2017 and 2019. These third-generation device models have similar width with XTR having arms that are one-third longer than NTR arms allowing for more leaflet tissue grasping. All patients were ≥18 years old and were deemed clinically and anatomically suitable for TEER with MC system by the heart valve team (comprised of an interventional cardiologist, a cardiothoracic surgeon, an interventional echocardiographer, and a heart failure specialist). All pre-procedural and patient-related data were prospectively collected, and consecutive patients having undergone successful implantation of at least one MC were included in the data analysis based on availability of perioperative 3D TEE volumes of the whole MV allowing evaluation of mitral annulus and leaflet anatomy, the MVA before clip (MVABC), and the MVA after one (MVA1MC) and two (MVA2MC) MC implantations. All measurements were based on 2D and 3D TEE data sets acquired before the intervention and after each individual MC implant. The LV end-diastolic volume, end-systolic volume, and ejection fraction were measured by means of the

Simpsons method of discs and using 3D volume sets when available.

In this study, MVA measurements were obtained using the "weaving" method developed by the authors rather than the "standard" multiplanar reconstruction (MPR) method. Using the "standard" method, MVA measurement is performed using MPR of MV 3D data set by trying to adjust a cutting plane in the LV at the distal borders of anterior and posterior leaflets to realize a direct planimetry. Although the "standard" method is most commonly used to measure MVA, it is based on the mistaken assumption that the margins of all the MV scallops are on the same plane during maximal diastolic opening. However, due to the funnel shape of the opened mitral valve, it is wider near the annulus and narrower in the left ventricle. This method has been validated in patients with mitral stenosis and tends to overestimate MVA in non-stenotic valves. To overcome this limitation of "standard" method, the authors developed a new "weaving" method to define the exact location of leaflet tips in several planes. Briefly, the edges of different MV leaflet scallops in the LV are marked on multiple planes perpendicular and parallel to the intercommisural diameter. These landmarks are then used to perform direct planimetry on the 3D data set. The "weaving" method should allow for more accurate measurement of MVA because it represents the 3D curvilinear surface of MV delineated by the actual position of the tips of the leaflets in 3D space by projecting the true tips of each aspect of each leaflet onto a single plane. Although the new "weaving" method takes into account the complex morphology of the MV, it has not been validated. The authors chose to use the "weaving" method for MVA measurement because it showed lower interobserver variability for MVABC, comparable value for MVA1MC, slightly smaller MVABC, and correlated better with the 3D volumetric MVA compared to the "standard" method.

The following 3D parameters were measured and/or calculated: MVA, annulus surface, intercommisural and antroposterior diameter of the annulus, sphericity index (anteroposterior/intercommissural diameter), anterior and posterior leaflets surfaces, and leaflet reserve (total leaflet surface divided by the annulus surface). After one MC implantation, the two created orifices were measured independently and the resulting MVA was calculated as the sum of both. The relative MVA reduction was calculated as 100 × ([MVABC – MVA1MC]/MVABC). Furthermore, the diastolic mean pressure gradient after one implant was calculated using the transmitral continuous wave Doppler velocity-time integral. Similar measurements were performed following the implantation of a second MC (MVA2MC). The following potential functional, 3D anatomical, and technical predictors of MVA after device implantation were evaluated: MR etiology, annulus and leaflet anatomy, model of MC used (NTR vs XTR), position of the device along the coaptation line, and, in the case of multiple implants, the distance between the clips.

Results

A total of 166 consecutive patients were included in the study. The median age was 81 years old, 42% were female, 47% had primary MR, and 94% had moderate to severe MR. An XTR clip was implanted as the first device in 50% of the patients, and 53% were treated with a single MC.

The median MVA reduction was 56%. Patients treated with an NTR had a lower relative MVA reduction than those treated with an XTR (52% ± 8 vs 57% ± 7%, P = .001). The percentage of MVA reduction was maximal for a noncommissural and noncentral location of the clip, which the authors refer to as "hot zone." Reduction in MVA did not differ according to MR etiology (primary vs secondary). In 26 patients, a second MC was placed close enough to the first one to create a double-orifice

MV, whereas in the remaining 28 patients there was a triple-orifice MV. After the second clip implantation, the incremental MVA decrease was 29% ± 10% for a total MVA reduction of 66% ± 7%. Patients with a triple-orifice MV had a higher MVA percentage reduction than those with a double-orifice morphology (34% ± 11% vs 25% ± 9%, P = .001). There was an inverse correlation between the percentage of MVA reduction and the leaflet reserve, suggesting that a lower ratio of leaflets to annular area may lead to higher tension and stabilization of the leaflets following device implantation.

The MVABC best correlated with post-procedural MVA. The model of clip used had no impact on absolute MVA1MC due to significant differences in MVABC between both groups (4.9 ± 1.3 for NTR vs 5.9 ± 1.7 cm2 for XTR, P = .003). Multivariate regression analysis identified MVABC, the model of MC, and the zone of implantation as predictors of MVA1MC (r = 0.91, P < .0001). The minimal MVABC needed for a single-device TEER ranged from 3.5 cm2 (one NTR in the ZoneCold) to 4.7 cm2 (one XTR in the ZoneHot). Among the 54 patients receiving a second MC, 22% had an MVA2MC < 1.5 cm2 (vs 12% after one clip) and 52% had a triple-orifice morphology. The mean MVA2MC was smaller in the case of triple-orifice morphology. Multivariate regression analysis identified MVABC, the implantation zone of the first device, and the morphology (double vs triple-orifice morphology) as predictors of MVA2MC. The minimal MVABC for a two-device TEER therapy varied between 4.5 cm2 (double-orifice morphology and first implant in the ZoneCold) and 6.3 cm2 (triple-orifice morphology with the first clip in the ZoneHot). The mean gradient was weakly inversely correlated with MVA1MC with pressure gradient of 3.6 mm Hg as the optimal cutoff to detect an MVA1MC < 1.5 cm2. The weak correlation can be explained by high interdependence of pressure gradients with hemodynamic parameters such as heart rate, stroke volume and residual MR.

Discussion

Although TEER is the most successful of the transcatheter mitral valve procedures and is a proven alternative to surgical mitral valve intervention in carefully selected patients, little is known about the predictors of MVA reduction after device implantation. The EVEREST anatomical criteria has been used to determine suitability for TEER with MVABC <4 cm2 deemed prohibitive.[4] However, a single cutoff value based on 2D echocardiographic measurements does not take into account complex anatomical and technical aspects of mitral valve edge-to-edge repair and bears the risk of excluding patients who would benefit from the therapy.

This is the first study to systematically review 3D MVA changes after each individual MC implant and model technical and anatomic parameters to predict the minimal MVABC needed to prevent clinically significant mitral stenosis. Furthermore, the authors first described and proposed the "weaving" method for 3D MVA measurement that enables tracking of the leaflet tips occurring in multiple planes. This new method addressed the limitation of "standard" method which is based on the MV leaflet tips projection on a single plane leading to inaccurate MVA assessment in the nonstenotic mitral valve. Finally, the authors derived a proposed algorithm for device and patient selection (Figure 1 next page).

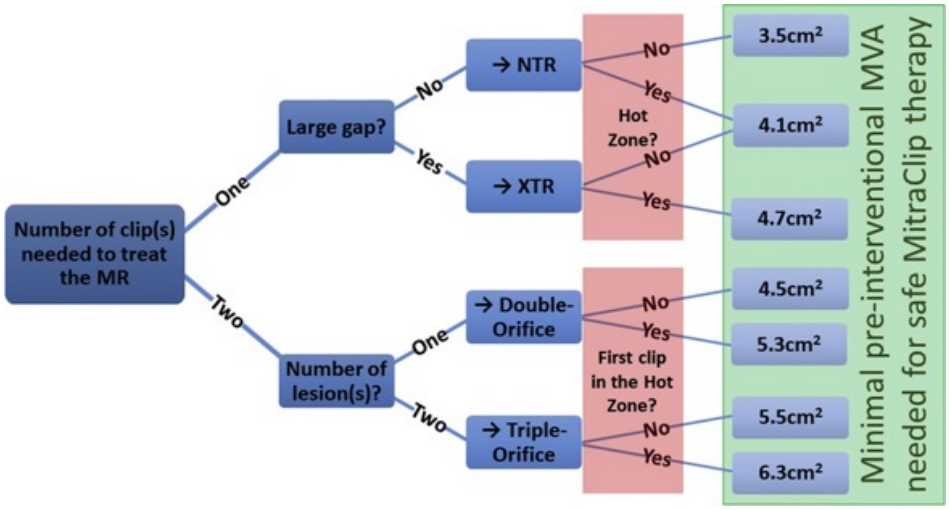

Figure 1. Algorithm for prediction of the lowest MVA needed before TEER according to anatomical parameters of the regurgitant lesion (borrowed from Kassar et al).

The authors determined several technical and anatomic parameters associated with MVA reduction after MC implantation(s). First, the final 3D MVA after MC implantation depends on the size of the native valve and on the model and position of the clip used. The implantation of one NTR or one XTR clip results in a mean MVA reduction of 52% and 57%, respectively. The increased relative MVA reduction with XRT device can be explained by higher tension of the MV produced by grasping of more leaflet tissue and increased leaflet stabilization. Second, the maximal MVA reduction occurs when the MC is implanted in a slightly eccentric position, referred to as "ZoneHot." MitraClip impairs opening of the valve at the locus of implantation and reduces the movement of the leaflets on both sides of the implant. The influence on the segments immediately adjacent to the device is maximal and decreases with the distance along the cooptation line. The authors postulate that the slightly eccentric position of MC in the ZoneHot results in greater MVA reduction due to increased leaflet tension when device is positioned where anterior MV is shorter (not A2/P2 region). Third, the implantation of a second MC adjacent to the first one reduces the MVA to a lesser degree than when a third orifice is created. This observation can also be explained by differences in leaflet tension and stabilization. Finally, the authors conclude that an arbitrary cutoff MVA ≥ 4.0 cm2 is not appropriate for all patients who are considered for TEER and does not reflect the complexity of the interactions between the valve and MitraClip. Some patients with an MVA before implantation of 3.5 cm2 can be treated with the implantation of one MC, while a minimal MVA of 4.5 cm2 is required for a two-MC strategy. Although mean transmitral pressure gradient can be used to predict MVAMC, interprocedural decisions should rely on direct 3D planimetry.

In addition to being performed at a single center, the study's limitations include small sample size and the use of non-validated methods for MVA assessment and prediction models. Although the absence of independent reference method for the MVA measurement before and after intervention does not allow validation of the "weaving" method, the models for MV prediction should be prospectively tested in a validation cohort. Furthermore, this study investigated patients treated with third generation MitraClip device models and applicability to fourth generation MitraClip device or wider models (NTW and XTW) is limited.

Despite these limitations, this study is a valuable contribution to the literature evaluating predictors of MVA reduction after TEER using MitraClip. Optimization of patient and device selection can not only improve patient outcomes but also expand patient eligibility.

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Anatomical and Technical Predictors of Three-Dimensional Mitral Valve Area Reduction After Transcatheter Edge-To-Edge Repair

Mohammad Kassar, MD, Fabien Praz, MD, Lukas Hunziker, MD, Thomas Pilgrim, MD, Stephan Windecker, MD, Christian Seiler, MD, and Nicolas Brugger, MD, Bern, Switzerland J Am Soc Echocardiogr 2022;35:96-104.

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Background

While several devices are being developed for percutaneous correction of severe mitral regurgitation (MR) the one most used is the MitraClip (MC). The repair resembles the surgical Alfieri stitch. The most common limitation of transcatheter edge to edge repair (TEER) for severe MR is the development of iatrogenic clinically significant mitral stenosis (MS) (as defined in 2017 guidelines1: 3D MVA<1.5 cm2). According to the current practice, If the pre procedure mitral valve area (MVA) is <4 cm2 by 2D echocardiography (EVEREST anatomical criteria2), TEER is usually not done because of the higher probability of development of clinically significant MS. The authors of this study are using 3D echocardiography measurements, to examine the factors (anatomical, functional or technical) that influence MVA reduction after TEER with one or more MCs. Using statistical analysis incorporating above measurements/ factors, they are estimating the minimal pre procedure MVA required to avoid clinically significant MS post procedure.

Methods

The study included echo recordings from 116 patients with symptomatic MR who underwent TEER with MC system NTR or XTR (Abbott Vascular, Abbott Park, IL) in Bern University Hospital, Switzerland, during 2017-2019. The 3D data sets were acquired peri-intervention with EPIQ7 or EPIQCVx with the X8-2t probe (Philips Healthcare, Eindhoven, The Netherlands) and were analyzed retrospectively.

LV ejection fraction (EF) measurements: 4D LV-ANALYSIS 3" module of TOMTEC-ARENA (TOMTEC Imaging Systems, Unterschleisshelm, Germany)3 or Simpson method of discs4 when 3D LV full volume data were not available.

MVA assessment before and after TEER: The "standard" and the "weaving" methods were used before the procedure (MVABC), after the first (MVA1MC) and after the second (MVA2MC) MCs. Because there is no gold standard for the measurement of MVA, particularly in the absence of MS, in patients with <1+ aortic regurgitation, the 3D volumetric MVA (LV stroke volume/continues wave doppler velocity time-integral inflow MV) was used as a reference.3 The "standard" method, validated for patients with MS5,6, is the direct planimetry of the MV orifice(s) when the cutting plane of the 3D multiplanar reconstruction (MPR) is positioned at the most distal edge of the MV leaflets or scallops. Because the actual orifice is a 3D multilevel structure as some segments reach deeper into the LV, the "weaving" method was developed to correct for the assumption of the "standard" method that MV orifice(s) is(are) in one plane. In the "weaving" method the "4D CARDIO-VIEW 3" module of TOMTEC-ARENA was

used which enabled detection of the tips of all leaflet segments in 3D space and by projecting all dots in a single plane produced a measurable orifice in one plane.

Evaluation of MV annulus anatomy: "4D MV-ASSESSMENT 2" module of TOMTEC-ARENA was used7 and the following parameters were measured: MVA, annulus surface, intercommissural and anteroposterior annular diameter, sphericity index (anteroposterior/intercommissural annular diameter), leaflet surfaces, and leaflet reserve (total leaflet surface/annulus surface).

Position of the first MC along the coaptation line: the area of the smallest neo-orifice as a percent of MVA1MC was calculated. A 50% would indicate central MC position and likewise a lower percent of the smallest orifice a commissural medial or lateral position.

Statistical analysis: Associations between anatomical MV parameters and MV area reduction after TEER were evaluated with Pearson correlation coefficients. Multivariate analysis of several factors as potential predictors of MVA reduction after TEER to MVA1MC or MVA2MC were evaluated: MR etiology, annulus and leaflet anatomy, iteration of MC used (NTR vs XTR), position of the MC along the coaptation line and the distance between the clips. The statistical software derived formulas permitting calculation of the predicted MVA1MC or MVA2MC. ROC (receiver operating curve) analysis was used to define the cutoff best predicting the development of MS.

Interobserver reproducibility and variability were assessed.

Results

Among the 116 studies included the median patient age was 81 years, 42% were female, an XTR MC was implanted first in 50% and 53% received 1MC.

The interobserver variability of MVA measurements was excellent but slightly better when measured with the weaving method. The measurements obtained with the "weaving" method were slightly smaller compared with the standard method and correlated better with the volumetric measurements, so the "weaving" method values were used for the analysis.

The etiology of MR did not affect the %MVA reduction.

The median %MVA reduction with 1XTR MC was 57%±7% vs 52%±8% with 1NTR with overall median reduction 56%. The greatest reduction in the MVA with 1MC was found when the MC was placed in the position 25-44% "ZoneHot" (non commissural and non central location) from the commissure with 50% been the middle of P2. Total %MVA reduction was 66%±7% when a second MC was used and with triple orifice morphology vs double orifice (additional reduction 34%±11% vs 25%±9%).

The MC type, clipping zone, intercommissural diameter and leaflet reserve were identified as predictors of %MVA reduction.

MVABC, the type of MC, and the implantation zone were identified as predictors of MVA1MC while MVABC, the implantation zone of the first device and the morphology of the neo MV orifice (double or triple) as predictors of the MVA2MC by multivariate regression analysis.

The minimal MVABC needed for safe 1 MC therapy ranges from 3.5 cm2 (1 NTR in ZoneCold) to 4.7 cm2 (1 XTR in ZoneHot), and for 2 MC therapy from 4.5 cm2 (double orifice morphology with first MC in the ZoneCold) to 6.3 cm2 (triple orifice morphology with the first MC in the ZoneHot).

The ROC analysis identified mean pressure gradient across the mitral valve of 3.6mmHg as the best cutoff value (sensitivity 86%, specificity 81%) to detect MVA1MC <1.5 cm2 but weakly inversely correlated with MVA1MC.

Discussion

This interesting study provides a systematic 3D echocardiographic evaluation of the changes in the MVA after TEER with 1 or 2 MCs in 116 patients. The authors summarize the results and propose an algorithm that can be used as a tool to predict post MC MVA and guide intervention in order to help decrease the incidence of iatrogenic MS.

In Summary

1. The final MVA after TEER depends on the type and position of the MC(s).
2. The XTR MC (9mm) results in greater MVA reduction than the NTR (6mm).
3. Maximal MVA reduction occurs in non-commissural and non-central MC positions.
4. The 3 orifice morphology results in a smaller MVA
5. The MVABC cutoff of 4 cm2 may not be appropriate for all patients: Some patients with MVABC of 3.5 cm2 can be safely treated with 1 MC while a minimum MVABC of 4.5 cm2 is required for the 2 MC intervention.

The proposed "weaving" method for MVA measurements has excellent correlation with reference values. The decision algorithm to minimize iatrogenic MS, constructed on ROC analysis has high sensitivity and negative predictive value. Both however were applied on a small single hospital cohort and have not been validated prospectively.

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Reintervention and Survival After Transcatheter Pulmonary Valve Replacement

McElhinney DB, MD, Yulin Zhang Y, Levi DS, et. al.
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Background

Transcatheter pulmonary valve (TPV) replacement (TPVR) first became utilized almost two decades ago. Now, PPVR has become the standard therapy for postoperative pulmonary outflow tract dysfunction in patients with an anatomically suitable prosthetic conduit/valve. Patients who receive TPVR usually have complex cardiac disease with a history of previous surgery, and present with right ventricular outflow tract obstruction or pulmonary valve regurgitation. However, there is limited information about short- and long-term outcomes, risk factors for death and/or reintervention after this procedure.

As with surgical traditional surgical conduits and valves, the 2 commercially available balloon-expandable TPV devices, the Melody valve (Medtronic Inc) and the Sapien valve (Edwards Lifesciences), can develop time-dependent dysfunction that merits reintervention. Also, patients with the underlying cardiovascular conditions most often treated with TPVR, including cono-truncal anomalies and left-heart diseases treated with a Ross procedure, can be prone to various adverse outcomes, including arrhythmias, dysfunction of other valves, and heart failure, and do not always have a normal life expectancy.

Study Design

This study was a multi-center cohort observational design that utilized an international registry for outcomes specifically pertaining to transcatheter after pulmonary valve replacement (TPVR). Data were submitted by investigators from 15 of 31 centers that were invited to participate; This study sought to evaluate mid- and long-term outcomes after TPVR in a large multicenter cohort.

Results

A total of 2,476 patients who underwent TPVR between July 2005 and March 2020 were included in the study, with 71 to 318 patients implanted at each center (median: 168 patients). Of these patients, 82% (n = 2,038) had a Melody valve implanted, and 18% (n = 438) received a Sapien valve. Patients ranged in age from 10 months to 79 years at implant (median: 20.5 years; excluding hybrid implants, the youngest patient was 3.1 years). Approximately 7% of the cohort was >= 50 years of age at implant, and 3% of patients were >= 60 years of age.

Follow-up

Patients were followed for a total of 8,475 patient-years. The median duration of follow-up among the entire cohort was 2.8 years. A total of 95 patients were known to have died after TPVR. Of these, 24 (25%) deaths were attributable to heart failure, 12 (13%) were related to an episode of endocarditis, 7 (7%) were related to complications of the TPVR procedure, 36 were from other known causes. The cumulative incidence of death was 8.9% -- 8 years after TPVR. On multivariable

analysis, age at TPVR, a prosthetic valve in other positions, and an existing transvenous pacemaker/implantable cardioverter-defibrillator were associated with death. The estimated survival over time amongst different age groups demonstrated that death was significantly shorter survival among patients who were older at TPVR. Estimated survival with an alternative age stratification, by decade, confirms the same trend among older adults.

TPV Reintervention. A total of 258 patients underwent reintervention on the TPV at a separate

procedure (ie, not including treatment of the pulmonary artery or conduit injury or placement of a second valve during the same catheterization). The initial reintervention was surgical pulmonary conduit/valve replacement in 136 patients (53%), valve-in-valve TPVR in 74 (29%), and balloon angioplasty of the TPV in 48 (19%). In general, the types of first TPV reintervention were proportionately similar across age groups, although older patients were relatively more likely to undergo transcatheter than surgical reintervention. At 8 years, the cumulative incidence of any TPV reintervention was 25.1% and of surgical TPV reintervention was 14.4%. Risk factors for surgical reintervention included age, prior endocarditis, TPVR into a stented bioprosthetic valve, and postimplant gradient.

Discussion

This multicenter registry compiled the largest series of patients treated with TPVR to date, was developed to study long-term outcomes. This study demonstrated that that survival and freedom from reintervention or surgery after TPVR are generally comparable to outcomes of surgical conduit/valve replacement across a wide age range. As is clear from population studies of survival over time in patients with Tetralogy of Fallot, pulmonary atresia, and complex congenital heart disease in general, these patients are at significant risk for premature death. The significance of comorbid factors such as presence of another prosthetic heart valve or of a permanent pacemaker or defibrillator, in addition to age, indicate that more medically complex patients, regardless of age, were at higher risk of dying.

The only major limitations of this study were those typically associated with a retrospective, longitudinal registry, use of cross-sectional data as independent variables, and no post-implant follow-up in 10% of patients. There was also no data gathered on TPV function over time. This is clearly a complex population that is at risk for premature death beyond procedural or reintervention-related mortality.

Related Reading

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PRO CON

PRO/CON DEBATE

The Fontan Patient

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Mortality rates for congenial heart disease has declined and survival has improved dramatically such that the life expectancy for most patients extends well into adulthood.[1] This has included congenital heart lesions for which biventricular function is not possible. In 1971 Francis Fontan and Eugene Baudet bypassed the RV by redirecting caval flows to the pulmonary arteries in three patients with tricuspid valve atresia.[2,3] The Fontan procedure is performed for a number of congenital cardiac conditions:

- Hypoplastic heart syndrome (Right and Left)
- Tricuspid atresia
- Pulmonary atresia
- Double inlet left ventricle
- Double-outlet right ventricle
- Unbalanced atrioventricular canal defects
- Ebstein anomalies (certain ones)

The Fontan procedure is a palliative treatment that is the culmination of several stages based on the premise that a 'subpulmonary ventricular pump is not compulsory for venous return to cross the pulmonary vascular bed.[4,5,6] Initial treatment for these patients include prostaglandins to keep the ductus arteriosus open, atrial septostomy, and/or performance of a systemic-pulmonary shunt. This is performed to improve pulmonary and/or systemic blood flow and oxygenation. Stage 1, the Norwood procedure, involves creating a neo-aorta to facilitate systemic blood flow, closure of the patent ductus arteriosus, and placement of systemic-pulmonary shunts, the latter including Blalock Taussig, Sano, or the Waterston shunt. In the second stage, a Bidirectional Glenn procedure redirects superior vena caval deoxygenated blood directly into the pulmonary arteries. Finally, the Fontan procedure is completed when the inferior vena cava is anastomosed directly into the pulmonary artery either via an external or internal shunt (FIGURE 1). In an earlier version, the right atrium (via the right atrial appendage) was anastomosed to the pulmonary artery. This Classic Fontan resulted in a dilated atrium, arrhythmias, protein-losing enteropathy, and progressive heart failure.[5,6,7,8] For all versions, caval flow passes directly to the low resistance pulmonary circulation without the contribution of a sub-pulmonary artery ventricle. Blood passes through the lung circulation and then the low pressure systemic or common atrium. A neo-aorta, which includes varying anatomical tissue contributions from the pulmonary artery and aorta directs oxygenated blood from the systemic ventricle to the rest of the body. Fontan procedures may include atrial septostomy or fenestration directing deoxygenated blood to the systemic atrium to relieve caval congestion (i.e. a 'pop-off valve') and improve preload to the systemic ventricle.5 While this might alleviate hemodynamic consequences of right sided congestion, it increases the occurrence of right to left shunt and hypoxemia. These stages are sequentially completed within 8-10 years of age. Long-term outcomes are worse for those whose Fontan procedure were completed after age 7 vs those by 4 years.[9] Later completion is associated with formation of aorto-pulmonary collaterals which increase right to left shunt.[9]

Three studies totaling nearly 2000 patients report survival up to 80% at 20, 30, and 40 following the Fontan procedure.[3,10,11] Although flow from the cava to the pulmonary arteries does not rely on a pulsatile chamber in between, systemic consequences occur due to a higher vena cava pressure and reduced cardiac output.[12] The occurrence of complications, the timing of heart transplantation, or death rises significantly by 35-40 years of age.[3,10,11] Only 53% of Fontan patients were NYHA Class 1 at 40 years of age and less than 40% were free of a serious adverse events including arrhythmias, right sided failure, thromboembolism, transplant and death.[3,10,11] Sinus node dysfunction occurs in up to 45% and atrial tachyarrhythmias occur in up to 60% of adult Fontan survivors and are causes of significant dysfunction and morbidity.[13,14] A systematic review of 6707 cases across multiple studies, reported 1000 deaths with a mean follow-up time of 8.2 years. Death was due to heart failure, arrhythmias, respiratory failure, renal failure, and hematologic complications.

Cardiac output depends on passive flow dependent on a pressure gradient driven by an elevated caval pressure, low pulmonary pressures/resistance, normal left heart function and the absence of any obstructions between the cava and the systemic chambers (FIGURE 2).[15] The systemic vasculature is connected in series to the pulmonary vasculature with a single pump to drive the blood forward.[6] For those with preserved contractility, the cardiac output is determined by loading conditions i.e., preload. At rest, the cardiac output of the Fontan patient is decreased to 70% compared to the non-Fontal patient.[12] Fontan patients develop a higher pressure caval pressures to 10-15 mmHg at rest to help drive blood forward. During exercise central venous pressures increase to > 15-20mmHg.[16,17,18,19] Fontan patients exhibit chronotropic incompetence and do not respond normally to stimuli. Inadequate heart rate and loss of atrio-ventricular synchrony compromise function and increase pulmonary venous pressure, pulmonary arterial pressures and eventually caval pressures. Even the most stable Fontan patients have a significant reduction in exercise capacity.[20]

The transpulmonary gradient, the difference between the central venous pressure and the systemic ventricular end-diastolic pressure, drives flow.[6,15] In the absence of a subpulmonary ventricle, higher central venous pressures are necessary to drive blood forward into the pulmonary circulatory system. The Fontan physiology system and elevated caval pressures results in reduced cardiac output and systemic blood pressure (FIGURE 2). Together, the lower systemic pressure and higher central venous pressure results in reduction perfusion pressure and venous congestion (FIGURE 2). Low pulmonary vascular resistance and adequate preload are crucial factors determining cardiac output. In addition, there cannot be obstructions in the Fontan circulatory system. For the patient with greater venous congestion and pressure and/or elevated pulmonary vascular resistance, preload to the systemic ventricle may be maintained by flow across a fenestration.[20]

Chronic venous pressure elevation puts patients at risk for systemic complications, pleural effusions, ascites, and end-organ dysfunction. Fontan patients experience of a number of dysfunctions and complications either due to the Fontan circulatory system or complications of the Fontan connections.[6] Circulatory failure, ventricular dysfunction, atrioventricular valve regurgitation, arrhythmia, heart block, coagulation disorders, liver and renal failure, protein losing enteropathy, and plastic bronchitis are potential complications of the Fontan circulation.[4,21]

With improvements in care, the likelihood that that Fontan patient will present for non-cardiac surgery requiring anesthesia has increased. A routine procedure in this population is the performance of esophagogastroduodenoscopy (EGD) to assess esophageal-gastric-intestinal pathology. Roughly 1/3 of Fontan patients will develop Fontan-associated liver disease (FALD) related to chronically elevated central venous pressures and decreased cardiac output.[22] Increased venous congestion decreases portal vein inflow, which is responsible for 70-80% of hepatic blood flow and 50% of oxygen delivery. Compensatory increases in hepatic artery flow is limited. Esophageal varices are a consequence necessitating regular surveillance.

The following Pro-Con addresses the anesthetic management of Fontan patients presenting for EGD: Monitored Anesthesia Care (MAC) or General Anesthesia with Positive Pressure Ventilation (GA)

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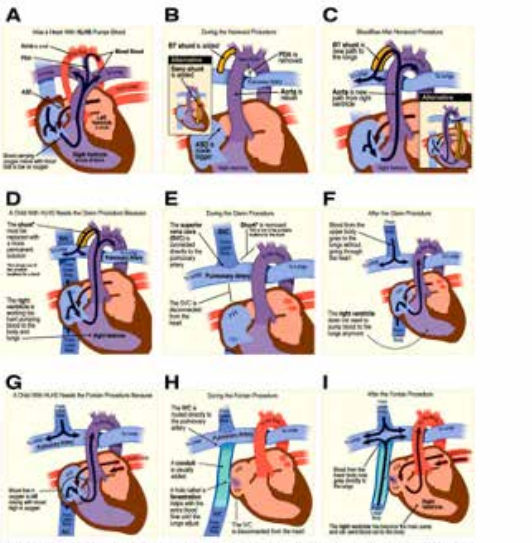
Figure 1.



Schematics showing the transition of a hypoplastic left heart syndrom (HLHS) to completion of a Fontan operation. A) HLHS: Small left heart and small aorta. Maintaing patency of the ductus arteriosus (PDA) and the presence of an atrial septal defect (ASD) are necessary for blood to be pumped into the systemic circulation, even if the blood is mixed oxygenated and deoxygenated. B and C) The Norwood procedure begins to separate the pulmonary circulation from the systemic circulation by directing blood into the pulmonary arteries by shunts (Blalock Taussig or Sano), and by creating a neo-aorta using whatever pulmonary artery and aorta are available typically combined with prosthetic material. The ASD is open to allow pulmonary vein flow to the systemic ventricle. The PDA is ligated. D, E, and F) The Glenn Procedure begins with the Norwood procedure (D) and directly connects the superior vena cava to the pulmonary artery. G, H, and I) The Fontan procedure begins after the Glenn procedure (G), and connects the inferior vena cava to the pulmonary artery by either an intra-atrial path or extra-cardiac (I) seen above. A connection, opening, or fenestration is noted (I) as a 'pop-off' valve to reduce venous pressure and congestion and increasing preload to the systemic atrium and ventricle.

Figure 2.

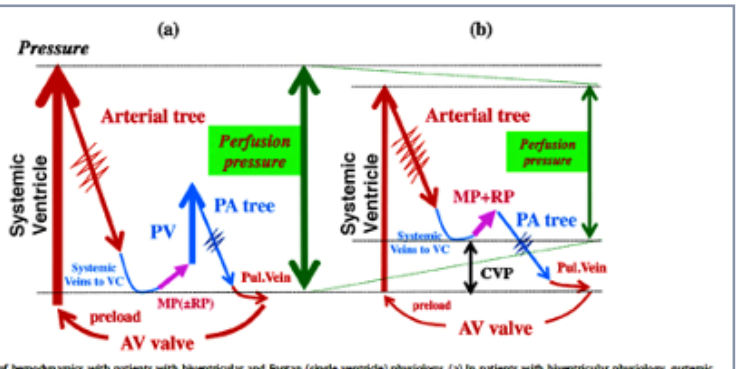


Fig. 2. Comparison of hemodynamics with patients with biventricular and Fontan (single ventricle) physiology. (a) In patients with biventricular physiology, systemic ventricle (SV) supports the systemic circulation and pulmonary ventricle (PV) supports the pulmonary circulation. Muscle pump (MP) has an additional impact on increase in cardiac preload (venous return). Perfusion pressure is well maintained. On the other hand, (b) in Fontan patients, SV supports the systemic circulation. High central venous pressure (CVP) is the driving pressure of the pulmonary circulation and the muscle and respiratory pumps (MP, RP) play significant role for the pulmonary circulation. Perfusion pressure is diminished due to high CVP and low systemic pressure. VC, superior/inferior vena cavae.

With permission from Hideo Ochuchi: Adult patients with Fontan circulation: What we know and how to manage adults with Fontan circulation? Journal of Cardiology 2016;68:181-189

PRO:

General Anesthesia for the Fontan Patient

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Patients with single ventricle physiology present a unique challenge to anesthesiologists. Fontan physiology is accompanied with reduced arterial oxygen saturation (Sa02) due to right to left shunting for which increasing fraction inspired oxygen (FI02) is only minimally effective.[1]

These shunts can be intracardiac resulting from a fenestration between the Fontan conduit and left atrium or from the coronary sinus draining into the left atrium. Or they can be intrapulmonary from reduced or absent hepatic derived venous factor in the cavopulmonary circulation.[2] Restrictive lung disease is also commonly present and too contributes to reduced Sa02.[3] Restrictive lung physiology decreases the time to desaturation with apnea or hypopnea, both of which are associated with administration of IV sedatives/anesthetics during sedation.

These common physiologic characteristics serve to make arterial oxygen desaturations a more likely event.

Equally central to this debate is the importance of maintaining low pulmonary vascular resistance (PVR).[4-6] CVP is the sole driving force for blood to pass through the lungs. Even minimal increases in PVR can be deleterious.[7] Pulmonary blood flow is not only essential for maintaining oxygenation, but it also provides preload to the systemic ventricle thus controlling cardiac output. Hypoxia and hypercarbia both increase PVR and decrease pulmonary blood flow which results in more still more hypoxemia .[8, 9] In a study of 15 pediatric patients, when arterial partial pressure of carbon dioxide (PaC02) was increased from 36mmHg to 55mHg, mean pulmonary artery pressures increased from 21 (+/- 6mmHg) to 30 (+/- 8mmHg) with an associated increase in PVR by almost 50%.[10] Another study of adult cardiac surgery patients demonstrated a 44% increase in PVR as C02 was increased from 30mmHg to 50mmHg.[11]

Although some Fontan patients may be able to tolerate these increases, there are also many for whom this increase would result in hypotension, increased shunting (particularly if a fenestration is present), hypoxemia and possible cardiovascular collapse.

All sedatives are associated with some degree of C02 retention including dexmedetomidine. Like other sedatives such as propofol, dexmedetomidine is associated with decreased hypoxic and hypercarbic respiratory drive.[12-14] Dexmedetomidine may seem an attractive adjuvant, but it can produce bradycardia which is poorly tolerated in Fontan patients due to their reduced single ventricle cardiac output.[15] While intravenous anesthetics have deleterious effects on ventilation, in a case series of 7 patients with pulmonary hypertension, anxiety was found to increase pulmonary artery mean pressure (mPAP) by 9 mmHg and PVR by 149 dyn * s *cm-5 as well.[16] This demonstrates the tightrope that must be walked to maintain adequate anxiolysis without deterioration of ventilatory status. IV sedation is associated with relative decreases in oxygen saturation which is also associated with increases in PVR. In a study of adult cardiac surgery patients with pulmonary hypertension, increasing FI02 to increase the Sa02 from 91 to 99% resulted in a decrease in PVR from 14.1 (+/- 1.4 WU) to 10.6 9+/- 1.0 WU).[17]

While the bulk of the discussion revolves around avoiding hypercarbia induced pulmonary vasoconstriction, its effects in Fontan patients can be questioned. We would be remiss to omit a study which introduces question into the effect of hypercarbia on PVR. In a study of 9 patients after bidirectional superior cavopulmonary anastomosis, postoperative ventilation was held constant and exogenous CO2 was added to inspired gasses to increase PaCO2 from 35 to 45 and then 55 mmHg. The results demonstrated that increasing PaCO2 from 35 to 45 mmHg improved systemic oxygenation, Qp, and Qs without increasing PVR.[18] While this might be cited as an example of the benign effects of hypercarbia on PVR, it should be noted that this study took place immediately post creation of a bidirectional superior cavopulmonary anastomosis in neonates and infants thus reducing its generalizability.

Aspiration or respiratory complications are a concern for sedated patients during endoscopy. The incidence of respiratory complications in a multicenter prospective observational study during endoscopy was 5.3% including coughing, fever, or shortness of breath with 0.1% requiring antibiotics.[19] Mortality for all comers who aspirate is between 1-4%.[20] In a retrospective study by Cooper et al, aspiration pneumonitis occurred in 0.1-0.14% of colonoscopies.[21] For most patients, aspiration and subsequent pneumonitis are manageable as there is sufficient cardiopulmonary reserve. However, in the single ventricle patient with already compromised pulmonary function, aspiration pneumonitis and pneumonia can be very detrimental. Increases in PVR, shunting, and decreased compliance are poorly tolerated and can result in substantial morbidity and mortality. Positive pressure ventilation if required can also be problematic.

Decreases in filling pressures from increased intrathoracic pressures can lead to diminished transpulmonary flow and decreased systemic ventricle preload.[8] Atelectasis associated with positive pressure ventilation can also increase shunting, leading to decreased oxygen saturation and increased PVR. In few instances the use of ECMO may be required which itself presents significant difficulties owing to alteration in anatomy and blood flow.[22]

Fontan physiology requires increased CVP for pulmonary flow which leads to venous engorgement of the head vessels and an SVC like syndrome.[23] Instrumentation of the airway in Fontan patients is associated with increased bleeding due to high these venous pressures. Use of oral or nasal airways can result in not inconsequential bleeding. In Fontan patients, placement and manipulation of nasal airways must be undertaken with great caution. The risk of substantial bleeding is further increased by the routine use of anticoagulants to prevent deep venous thrombosis (DVT) or Fontan thrombosis in this population.[23, 24] Warfarin is the most employed agent and if bleeding does occur from oral or nasal airway devices/ manipulation, aggressive management strategies which include PCCs can increase the risk of DVT or Fontan thrombosis. Although four factor purified prothrombin complex does contain protein C and S, the half-life of thrombin is substantially longer contributing to delayed hypercoagulability.[25] These procoagulant substances should be used sparingly in Fontan patients for this reason.

Lastly, obstructing and gaging from secretions can result in Valsalva which can further reduce filling pressures to the heart. Gaging and obstructing can also exacerbate hypoxemia. These occurrences together can result in increased PVR and decreases in transpulmonary blood flow and preload to the heart. Laryngospasm during sedation also possible. In the patient with biventricular circulation and minimal pulmonary compromise, this is usually tolerated and can be broken with gentle positive pressure or administration of a small dose of anesthetic. But in the patient with Fontan circulation, laryngospasm can result in hypoxemia and impairment of transpulmonary blood flow. Since Fontan patients already have some degree of pulmonary shunt and restrictive lung disease, the deleterious effects of laryngospasm are amplified. Negative pressure pulmonary edema would be poorly tolerated in this population as well.[26]

General anesthesia can be induced in a controlled and measured manner. With paralysis, patient movement, bucking and inadequate ventilation can be avoided. Passage of the endoscope is facilitated with relaxation and the operative field is motionless. Fontan

associated liver disease is a well-described phenomenon. The pathophysiology is thought to be related to liver injury secondary to passive hepatic congestion, and a component of hypoxic hepatopathy secondary to low-cardiac output states. The endoscopic banding and cauterization of these lesions can be challenging. GA is associated with a more stationary surgical field.

In conclusion, complications from IV sedation in patients with normal biventricular physiology are easily treated by simply repositioning of the airway, provision of brief positive pressure face mask ventilation, conversion to general anesthesia, or even cancellation and rescheduling of the procedure. Morbidity in these instances is extremely low; hypoxemia, hypercarbia, even aspiration rarely result in serious consequences. But these occurrences should be actively avoided in the Fontan patient, lacking the right ventricle to assist pulmonary blood flow. Fontan patients are on a time limited cardiac course; all are on the continuum of failing.[27] These adverse events can expedite this time course. While requiring induction and intubation, GETA removes these risks thus tipping the balance in favor of general anesthesia.

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CON:

MAC for Fontan Patients Undergoing EGD is Preferred

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Perioperative complications occur in 30-40% of Fontan patients undergoing noncardiac surgery.[1,2,3] Esophago-gastro-duodenoscopy (EGD) for non-Fontan patients is commonly performed under moderate or deep sedation. There is little consensus as to the choice of anesthetic for this seemingly low-risk procedure in a Fontan-patient with single ventricle physiology. Whether MAC with spontaneous negative pressure ventilation or GA with positive pressure ventilation is planned, the same basic principles of care apply:

1. Avoid increases in pulmonary artery pressures and/or pulmonary vascular resistance
2. Avoid reductions in preload
3. Avoid arrhythmias
4. Avoid depression of systemic ventricular function
5. Avoid high airway pressures, high PEEP, high tidal volumes, and long inspiratory time

Given what is known about the Fontan physiology; cardiac depression, increases in PVR and PAP, and reductions in cardiac preload are detrimental. Described pulmonary hypertension triggers include:

1. Hypoxemia
2. Hypercarbia
3. Acidosis
4. Hypothermia
5. Sympathetic stimulation
6. Pain, shivering, and anxiety

The choice of anesthesia depends on the known effects of anesthetic agents, the anticipated duration and invasiveness of the procedure, the baseline cardiopulmonary function, and the function of end-organs.[4] Sedation is less likely to be adequate for more invasive procedures with longer durations. However, for a surveillance EGD, general anesthesia has a high risk of compromising the patient with Fontan physiology.[1] The determinants of the Fontan circulation and systemic blood flow are multiple. These include systemic venous pressure, venous capacitance, pulmonary vascular resistance and pressures, cardiac rhythm, systemic ventricular systolic and diastolic function. Parameters are all made worse with the cardiovascular depressant effects of general anesthetics and volatile agents.[5]

The effects of general anesthesia with positive pressure ventilation on the cardiopulmonary system places the Fontan patient at risk for cardiopulmonary adverse events. With the exception of Etomidate, all general anesthetics depress ventricular function, dilate venous beds and reduce cardiac preload. General anesthesia also lowers systemic vascular resistance and coronary flow.[6,7,8,9,10] Desflurane, Nitrous Oxide and Ketamine (in adults) increase pulmonary vascular resistance (PVR).[6,7,8,9,10,11]

The stable, well-oxygenated Fontan patient with venous pressures < 10-12 mmHg, normal PVR and normal systemic ventricular function will tolerate general anesthesia or sedation.[1,12] For the more compromised patient, sedation will avoid the toxic effects of general anesthesia and positive pressure ventilation.[13] Reduction in caval pressures and volume due to the dilating effects of general anesthesia, positive pressure ventilation, and increases in PVR could be devastating to the Fontan circulation.[13] Any dysfunction of the systemic ventricle whether it be contractile dysfunction, relaxation abnormalities, and/or valvular dysfunction will further compromise the Fontan circulation.[13]

In addition to the depressant effects of general anesthesia, positive pressure mechanical ventilation is associated with pulmonary dysfunction and increased PVR. There is a U-shaped or bimodal relationship between lung volume and PVR.[14,15] Atelectasis, which occurs in up to 90% of patients undergoing general anesthesia, results in alveolar collapse, regional hypoxic pulmonary vasoconstriction of extra-alveolar vessels and increased PVR.6,7,8,9,10,11 While meant to minimize atelectasis, positive pressure ventilation with or without positive end expiratory pressure (PEEP) also increases PVR by alveolar overdistention and alveolar vessel compression.[8,14,16] Management of mechanical ventilation requires a balance between maintaining lower airway pressures and efforts to prevent atelectasis. When possible, spontaneous ventilation with normoxia and normocarbia has the least impact on PVR.[12] Increased PVR with positive pressure ventilation hinders passive blood flow requiring adjustments to minimize airway pressures to < 20 cmH2O, which then risks atelectasis and elevations in PVR. Recruitment breaths would only add to increases in PVR.[12] A transition from general anesthesia with PPV to spontaneous ventilation can be associated with acute changes in oxygenation and ventilation and the development of hypoxemia and hypercarbia highlighted by confusion, tachycardia, tachypnea, coughing, and hypoventilation.[3] Alternatively the patient may be allowed to breath spontaneously via an ETT with avoidance of muscle relaxation. However, to tolerate the ETT greater anesthetic concentrations may be required, associated with increased cardiovascular depression. At least 50% of complications occur in the postoperative period. Relatively simple issues such as nausea and vomiting, and shivering, will have more pronounced effects for the Fontan patient.[17] Each of these have a greater occurrence with increased dose of general anesthesia.1 A return toward baseline cardiopulmonary functions are complicated by residual anesthetic and a slower return to normal breathing patterns.

When the surgical procedure permits, sedation with spontaneous negative pressure ventilation is preferred. Spontaneous ventilation removes the hemodynamic effects of positive pressure ventilation and eliminates the need for transitions. Although a U-shaped relationship between lung volume and PVR has been described based on data from the 1960s, the details of these studies reveal greater compromise with larger lung volumes. While atelectasis causes compression of larger arteries, positive pressure inflation compresses smaller arteries, the latter of which are more significant contributors to pulmonary vascular resistance.[18,19] Under general anesthesia the likelihood of larger lung volumes compressing small vessels and the occurrence of atelectasis are significant.[18,19] Compared to spontaneous breathing (which may also be associated with atelectasis), the changes in pulmonary vascular resistance are significantly higher with positive pressure ventilation and higher lung volumes.[18,19]

The pulmonary vasculature is reactive and can change quickly in the presence of vasoconstrictors and/or the physiologic effects of hypoxemia and/or hypercarbia.1 Under normal conditions, the contracting RV may compensate and maintain normal coupling between the RV and the PA. However, in the absence of a sub-pulmonic contracting ventricle, pulmonary blood flow becomes more vulnerable to these physiologic changes. Subsequently, preload to the systemic ventricles drops, and systemic cardiac output declines.[1,20]

Sedation cases are associated with hypercarbic acidosis, hypoxemia, atelectasis, cardiac depression, and systemic vasodilation.[7,8,21] The effect of CO2 on pulmonary vascular tone is not straightforward with data showing both vasoconstriction and vasodilatory effects depending on the associated metabolic and physiologic conditions.[22] Under normal vascular tone, CO2 causes a weak vasoconstriction made worse under acidic conditions and attenuated during alkalosis.[23,24,25] The increased pulmonary vascular tone due to hypercarbia occurs over minutes but plateaus after 10-15 minutes.[26,27] Interestingly, hypercapneic acidosis may have beneficial effects on gas exchange by improving ventilation-perfusion matching via hypoxic pulmonary vasoconstriction.[27] In an animal model, hypercapnia over 100 minutes increased pulmonary artery pressures 2-3 mmHg and was associated with less pulmonary shunt, improved oxygenation and gas exchange, and less tissue edema.[27] By contrast, a vasodilatory effect reported with hypercarbia was seen in the presence of higher pulmonary artery pressures, or under conditions that induce vasoconstriction such as increased catecholamines, high endothelin-1 levels, or hypoxia with hypoxic pulmonary vasoconstriction.[22]

Hemodynamics, cardiac function, systemic blood flow, and oxygen delivery may be improved during hypercapneic conditions.[28] Although hypoventilation induced hypercapnia increased peak pulmonary artery pressure from 23 to 27mmHg in the first hour and then to 31 mmHg by 4 hours it was associated with greater cardiac output by increasing heart rate.[29] By comparison, hyperventilation induced hypocapnia reduced peak PAP from 23 to 18 in the first hour and to 16 by four hours but also reduced cardiac output by reducing stroke volume.29 During video assisted thoracic surgery, a lower respiratory rate causing hypercarbia for 30 minutes increased oxygen content and delivery in association with improved pulmonary mechanics, lower peak and plateau airway pressures, and improved pulmonary compliance.[30] Compared to normocarbia there was no difference in central venous pressure.[30]

The absence of detrimental effects and potentially beneficial effects of hypercarbia are also reported in pediatric patients with Fontan physiology.[31,32,33] Systemic oxygen delivery and cerebral blood is improved with hypercarbia. In 12 intubated ventilated patients with superior cavopulmonary connections; the effects of room air, hyperoxia and hypercarbia (inhaled CO2) were studied.[31] With PCO2 raised between 48-63 mmHg blood flow to brain increased 1.5 to 2.7 L/mn/m2. Under these conditions oxygenation and cardiac index increased significantly.[31] By contrast, in six patients after completion of a bidirectional superior cavopulmonary connection hyperventilation was shown to reduce arterial PO2, systemic oxygen saturation, and cerebral blood flow as measured by middle cerebral Doppler.[32] In a prospective study of 15 pediatric patients s/p bidirectional superior cavopulmonary connection, the effect of hypoventilation and hypercarbia were studied in the intensive care unit within 8 hours of surgery while sedated, paralyzed and mechanically ventilated.[33] Hypoventilation induced hypercarbia (PCO2 58 mm Hg) was compared to normocarbic ventilation. Despite small increases in pulmonary artery pressures and transpulmonary gradient, hypercarbia was associated with significant increases in mean PaO2 from 50 mm Hg at baseline to 61 mm Hg, mean SaO2 from 86% at baseline to 90%, and cerebral blood flow from 37 cm/s to 55cm/s.[32,33]

After bidirectional superior cavopulmonary connection hemodynamic performance improves with hypercarbia.[34] In 9 patients an increased CO2 from 35 to 55 was associated with increased PaO2, systemic and pulmonary blood flow, and cerebral blood flow.[34] Systemic vascular resistance declined and pulmonary vascular resistance remained unchanged or insignificantly reduced.[34] The authors concluded that that 'hypoxemia after the bidirectional superior cavopulmonary is ameliorated by a higher PaCO2 and that low PaCO2 or alkalosis may be detrimental'.[34] Hypercarbic management strategies may 'reduce interval morbidity in patients with a functional single ventricle'.34 During this study, there were insignificant increases (1-2 mmHg) in caval pressure even with a reduction in pH from 7.43 to 7.28 (PCO2 55 mmHg).[34]

Comparative hemodynamic and pulmonary functions in Fontan patients are seen during the transition from positive pressure ventilation to negative pressure spontaneous ventilation.[13] With pulmonary blood flow occurring passively, a lower pulmonary vascular resistance and lower intrathoracic pressures will improve flow and ultimately systemic cardiac output. By contrast, during positive pressure ventilation, increasing intrathoracic pressures by increasing PEEP from 3,6,9, to 12 cmH2O, increased PVR and reduced preload.[13] Spontaneous inspiration increased venous return and pulmonary blood flow 3-4 fold with a significant contribution from the liver.[13] The greatest benefit of spontaneous breathing was demonstrated in those that were less stable with lower cardiac outputs and hypoxemia.[13] While recognizing that hypercarbia may cause pulmonary vascular constriction, these effects are countered by lower intrathoracic pressures, improved pulmonary blood flow, cardiac output, oxygen content and delivery, even with PCO2 as high as 55-65mmHg.[13]

For the Fontan patient the systemic and pulmonary circulations exist in series driven by a single ventricular pump. In the absence of a subpulmonary ventricle flow from the cava to the pulmonary artery is passive being dependent on a pressure gradient between the systemic ventricle to the pulmonary artery, which is impacted on by central venous pressure, pulmonary artery pressure, and intrathoracic pressure.[20] The impact of heart-lung interactions, or cardiorespiratory coupling, on hemodynamics are multiplied in the Fontan patient. During spontaneous ventilation the contracting diaphragm acts as a 'pump', creating a negative gradient, drawing blood into the pulmonary vessels.[20] In the Fontan patient spontaneous inspiration increase venous return by 35%, while expiration increases aortic outflow by 30%.[20] In the Fontan patient positive pressure ventilation impairs venous return and cardiac output, while spontaneous negative pressure ventilation enhances it.[20]

Concerns of atelectasis and hypercarbia inherent to MAC anesthesia are countered with the hemodynamic benefits and increased oxygen content and delivery reported in the Fontan patient. During an EGD, when conditions permit it, sedation with spontaneous ventilation is preferred to harmful hemodynamic effects of general anesthesia, endotracheal intubation and positive pressure ventilation. Ultimately, the anesthetic management should be individually designed for each patient and depend on the baseline function of the Fontan circulation to prevent depression of the systemic ventricle, harmful increases in pulmonary pressures and resistances, and reductions in cardiac preload.[4] However, whether it be an asymptomatic acyanotic patient or one who is more significantly compromised; minimizing anesthetic concentration and maintaining spontaneous ventilation is beneficial for cardiopulmonary function.[4,13]

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