SCA NEWS

SOCIETY OF CARDIOVASCULAR ANESTHESIOLOGISTS
Knowledge • Care • Investigation

Volume 31, Number 15
June 2022

2022 TAS, COR-PM AND ANNUAL MEETING

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

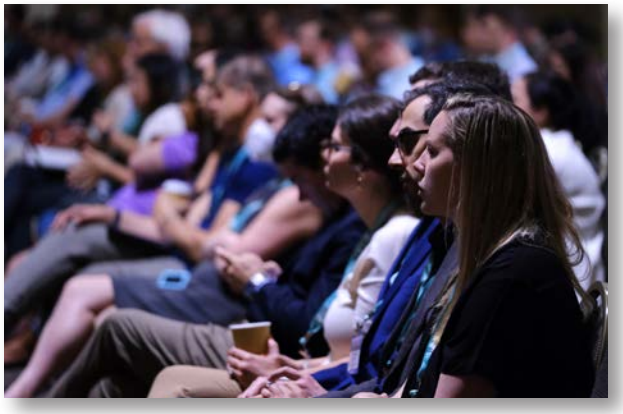
Thank you to all who attended in-person and virtually!

On behalf of the SCA Leadership and the Program Planning Committees, THANK YOU to the attendees and faculty for making the 2022 Meetings such a huge success! We hope you enjoyed the meetings as much as we did!

Continued...

2022

2022 TAS, COR-PM AND ANNUAL MEETING



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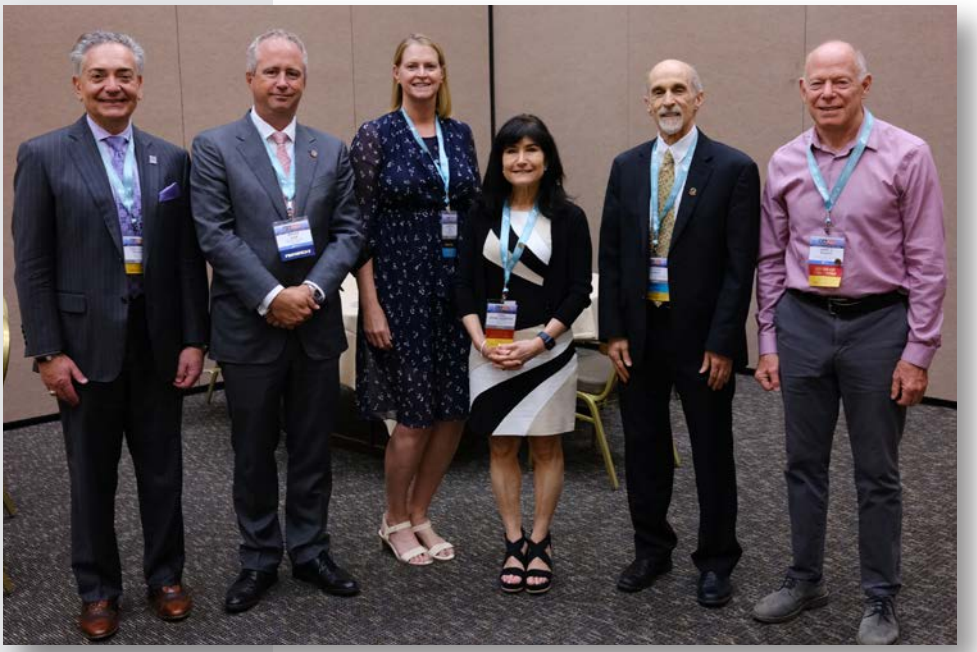
2022 TAS, COR-PM AND ANNUAL MEETING



Do Not Forget to Claim Your CME!

Meeting attendees - have you claimed your CME credits yet?

CME credits are available for TAS and COR-PM until July 12, 2022 and July 18, 2022, for Annual Meeting.

SAVE THE DATE
PERIOPERATIVE ULTRASOUND COURSE

SCA PoCUS 2023

SCA PoCUS Planning Committee invites you to join us in Atlanta, GA on February 16, 2023, at the Loews Atlanta Hotel to enhance your ultrasound skills. Registration is scheduled to open in early fall.

Watch your email for more details in the coming months.

PoCUS 2023

SCA ECHO WEEK 2023

Echo Week will be held February 17-19, 2023, in Atlanta, GA, at the Loews Atlanta Hotel.

Join us in Atlanta, GA, to meet, learn from, and connect with cardiovascular anesthesiologists from around the world. Registration is scheduled to open in early fall.

Watch your email for more details in the coming months.

ECHO WEEK 2023

SCA COR-PM 2023

Join us for the 2nd Annual Cardiovascular Outcomes Research in Perioperative Medicine (COR-PM) conference to be held May 5th, 2023, in Portland, Oregon.

More details will be forthcoming in the coming months.

COR-PM 2023

SCA Thoracic Anesthesia Symposium & Workshops 2023

SCA and the TAS Planning Committee will hold its Annual Thoracic Anesthesia Symposium & Workshops in Portland, Oregon, May 5, 2023.

Mark your calendars for this is a one day event focused entirely on thoracic anesthesia for academics and private practitioners.

More details will be forthcoming in the coming months.

TAS Abstracts – Here's Your Chance to Present

You are invited to submit a scientific abstract or complex case for consideration for the 2023 Thoracic Anesthesia Symposium & Workshops!

The SCA website will be updated as more information becomes available.

Call opens September 2022

TAS 2023

SCA Annual Meeting 2023

SCA and the Scientific Program Committee invite you to join us in Portland, Oregon for the 45th Annual Meeting and Workshops, May 6-9, 2023.

Mark your calendar NOW to join us for the 45th Annual Meeting and Workshops in Portland, OR.

More details will be forthcoming in the coming months.

PBLD Submissions for the 2023 Annual Meeting Open Soon!

Submit your 2023 Problem Based Learning Discussion for the Annual Meeting & Workshops.

When submitting a PBLD, you will be asked to provide the following information:

- Primary Moderator Information*
- Co-Moderator Information (optional)
  - Name
  - Email
- PBLD Title*
- Session Objectives*
- Overview of the Case Presentation*
- Case Questions (optional)

Call opens July 2023

Only one submission per person will be considered.

The SCA website will be updated as more information becomes available.

Submit an Abstract for the 2023 Annual Meeting & Workshops!

Get ready to submit your scientific abstract or complex case to be considered for presentation at the 2023 Annual Meeting & Workshops!

Submissions will be accepted for the following calls:

- Scientific Program
- Fellow and Resident Complex Cases
- Super Echo

The SCA website will be updated as more information becomes available.

Call opens September 2023

SCA 2023

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 toward improving our society, and we thank them for their involvement.

Founding Officer Successor, 2010-2022

Glenn P. Gravlee, MD
University of Colorado Denver

Board Director 2021-2022

Michael P. Eaton, MD
University of Rochester School of Medicine

Chair, Scientific Program Committee, 2020-2022

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

Early Career Director 2020-2022

Emily Methangkool, MD, MPH
UCLA

EACTAINC Liaison 2020-2021

Gianluca Paternoster, MD

CME Committee 2018-2022

Jennifer Hargrave, DO
Cleveland Clinic

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Meet SCA's 2022-2023 New Board of Directors

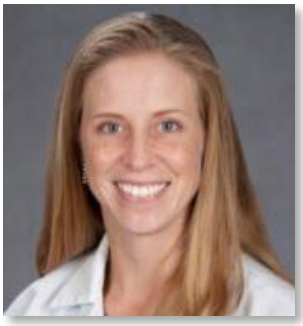
Board Director (Re-elected) 2022-2025

Annemarie Thompson, MD
Duke University

Board Director 2022-2025

Daryl Oakes, MD
Stanford University

Early Career Director (Re-elected), 2022-2024



Jessica Brodt, MD
Stanford University

Early Career Director 2022-2024

Stephanie Ibekwe, MD
Baylor College of Medicine

Founding Officer Successor, 2022-2028

Linda Shore-Lesserson, MD, FAHA, FASE
Northwell Health

Chair, Scientific Program Committee 2022-2024

Mary Beth Brady, MD, FASE
Johns Hopkins University School of Medicine

Vice-Chair, Scientific Program Committee 2022-2024

Jonathan K. Ho, MD, FASE
University of California – Los Angeles

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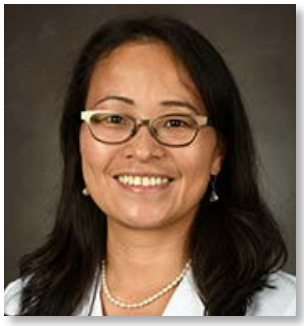
2022 Award Recipients

SCA is excited to announce the following 2022 grant and award winners.

SCA/IARS Mid-Career Research Grant

$50,000 per year for 2 years



Yafen Liang, MD
Associate Professor
McGovern Medical School, UT Health Science Center

Grant Title: *Prevention of Post-Cardiac Surgery Acute Kidney Injury by Proton Pump Inhibitor: A Prospective Randomized Controlled Trial*

SCA/IARS Starter Research Grants

$25,000 per year for 2 years

Meghan Prin, MD, MS
Assistant Professor
University of Colorado Denver

Grant Title: *Circadian Movements and Delirium in Older Cardiac Surgery ICU Patients*

SCA/IARS Starter/Diversity Research Grants

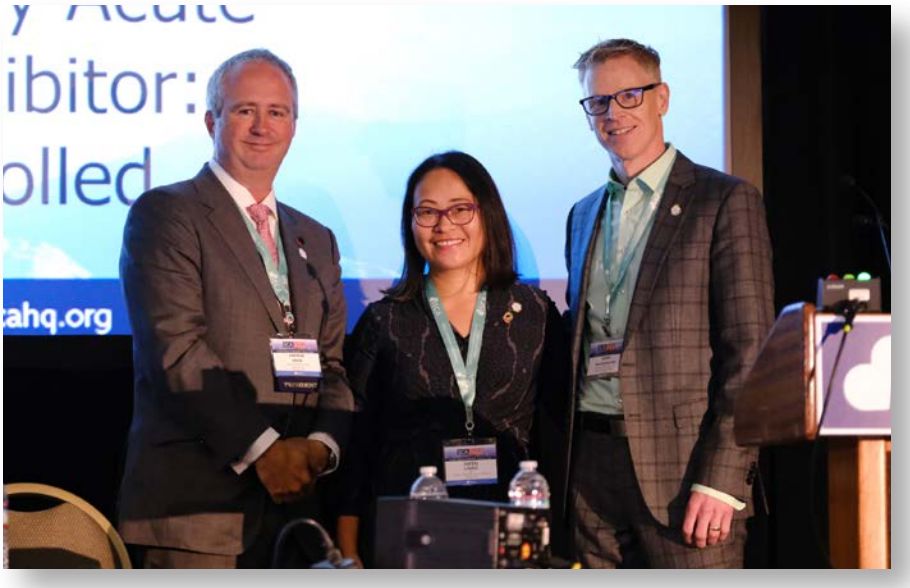
$25,000 per year for 2 years

Kofi Vandyck, MD
Assistant Professor
University of Oklahoma Health Science

Grant Title: *Optimization of Platelet Function Testing In Cardiopulmonary bypass (OPTIC): A Comparison Between Thrombelastography-Platelet Mapping and Total Thrombus-formation Analysis System (T-TAS)*

Congratulations to SCA's 2022 Grant Recipients!

(Left to Right) Drs. Shaw, Vandyck and Muehlschlegel



(Left to Right) Drs. Shaw, Liang and Muehlschlegel

SCA Kaplan Leadership Development Award Winners

Sheela Pai Cole, MD, FASA, FASE
Clinical Professor
Stanford University

Leadership Project Title: *Stanford LEAD Program (Learn, Engage, Accelerate, Disrupt)*

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

Leadership Project Title: *University of Texas at Dallas Executive MBA – Healthcare Organizational Leadership*

Congratulations to the 2022 SCA Kaplan Leadership Development Award Recipients!

(Left to Right) Drs. Shaw, Ibekwe, Pai Cole and Perry

SCA 2022 Early Career Investigator Award Winners

Lauren Gibson, MD
Massachusetts General Hospital

Abstract Title: *Post-Cardiotomy Right Ventricular Strain Predicts the Need for Rescue Venoarterial Extracorporeal Membrane Oxygenation*

Daryl Kerr, MD
Duke University

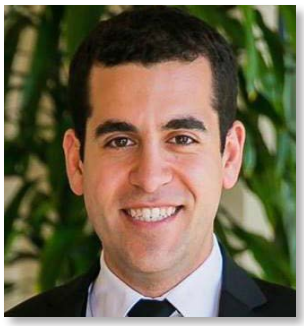
Abstract Title: *Preoperative Right Ventricular Dysfunction Severity and Cardiac Surgery Outcomes (RV DYSCO) - an analysis of the Society of Thoracic Surgeons Database*

Rosa Kim, MD, MS
Tufts Medical Center

Abstract Title: *Comparison between Erector Spinae Plane Blocks (ESPB) and Transversus Thoracic Plane Blocks (TTPB) in cardiac surgery patients: a retrospective observational study*

Michael Li, MBBS, MPH
Duke University

Abstract Title: *Use of intravenous fat emulsion to suppress 18F-fluorodeoxyglucose uptake in non-ischemic myocardium for cardiac positron emission tomography*



Nabil Thalji, MD, PhD
University of Pennsylvania

Abstract Title: *Characterization of Andexanet-Associated Heparin Resistance: Implications for Management*

2022 Distinguished Service Award Winner

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

Joyce Wahr, MD
University of Minnesota

Congratulations Dr. Wahr!

(Left to Right) Drs. Shaw and Wahr

2022 Presidential Lifetime Outstanding Service Award Winner

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

Bruce Spiess
University of Florida

Congratulations Dr. Spiess!

(Left to Right) Drs. Shaw and Spiess

MEMBER CORNER

STILL AVAILABLE!

2022 TAS and Annual Meeting Online Content

The Thoracic Anesthesia Symposium and SCA 2022 Annual Meeting and Workshop lectures are still available for purchase for the next six months. If you have not already purchased the content, click here to start your process.

SCA's Presidential Historical Taskforce

SCA will be celebrating its 45th Anniversary in 2023! A Presidential Historical Task Force has been established to collect, preserve, and share the rich history of the Society with current and future members. The Task Force is being led by long time SCA member and former President, Glenn Gravlee.

The Task Force is asking those of you who may have SCA related memorabilia, documents, photos, old equipment and/or great stories to share to please reach out to Jim Pavletich at SCA. We are very interested to learn of these items and to find ways to incorporate them into the final work product(s) of the History Task Force.

Please share any item(s) by July 31, 2022 – to allow the Task Force to collectively review, categorize and decide whether/how they may be presented.

The Call for Nominations for the SCA Board of Directors

Show your commitment to the value of the Society of Cardiovascular Anesthesiologists to shape its future! You may nominate yourself or a committed SCA colleague. This year we will elect two Directors-at-Large,
a President-Elect, and a Secretary/Treasurer.

Opening in August 2022!

We will also be electing two members to the Nominating Committee, and one member to the Continuing Education Committee (CME). These committees are elected positions.

More details are forthcoming in the next month.

The Call for Volunteers is Moving!

The Call for Volunteers, which is the process in which SCA committees are populated, will now take place in October for the 2023-2024 term.

Previously, this process occurred around January. Watch your in-box and future issues of the Newsletter for more details.

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Coming Shortly - New Research Funding Available!

The Call for PUF Research Grant Proposals is scheduled to open in July 2022. Each selected applicant will be awarded up to $15,000 to apply for and complete an STS Participant User File (PUF) Grant.

More information on how to apply will be available in the coming weeks!

Requirements for proposed research projects are available at **https://www.sts.org/research-center/programs-and-data-access/participant-user-file-research-program**.

Find more information on the use of the Adult Cardiac Section of the STS Adult Cardiac Surgery Database at **https://www.jcvaonline.com/article/S1053-0770(20)30798-9/pdf**

Support Your Society through the SCA Endowment

SCA is the preeminent international educational organization for this sub-specialty, leading the way in treatment innovations through care, investigation, and knowledge. By donating to the SCA Endowment, the funds help support SCA professionals to further their education, research, and professional development and to achieve their goals.

The SCA Endowment Fund online donation page is available. Making an online donation is quick, easy, and secure. To complete the online donation form, visit **https://scahq.org/about/sca-endowment/**.

Vision of the SCA Endowment

We will be world leaders in enhancing patient care and safety and in developing excellence in the next generation of clinicians and physician-scholars through research and education in the field of cardiothoracic and vascular anesthesia.

For more details on the endowment, please email donation@scahq.org.

THORACIC CORNER

Characteristics and outcomes of patients with COVID-19 supported by extracorporeal membrane oxygenation: A retrospective multicenter study

Saeed O, Tatooles AJ, Farooq M, et. al. Characteristics and outcomes of patients with COVID-19 supported by extracorporeal membrane oxygenation: A retrospective multicenter study. J Thorac Cardiovasc Surg. 2022 Jun;163(6):2107-2116.e6. doi: 10.1016/j.jtcvs.2021.04.089. Epub 2021 May 18. PMID: 34112505; PMCID: PMC8130603.

Reviewers:

Melissa Burtoft, MD
Division of Anesthesiology and Perioperative Medicine,
Mayo Clinic, Rochester, MN

Ashley Fritz, DO
Division of Cardiovascular and Thoracic Anesthesiology,
Mayo Clinic, Jacksonville, FL

Background

The treatment for acute respiratory distress syndrome (ARDS) has evolved over the last two decades to include extracorporeal membrane oxygenation (ECMO) in cases of refractory lung injury[1]. In addition to low tidal volumes, high positive end-expiratory pressure, neuromuscular blocking drugs, and prone positioning, ECMO further reduces lung injury in ARDS by allowing for more gentle lung ventilation while providing complete oxygenation and ventilation[2]. The COVID-19 pandemic led to increased incidence of severe lung injury and ARDS requiring advanced therapies and the increased need for rescue ECMO therapy[3]. As the pandemic continues to evolve, there is still little evidence on outcomes of patients with COIVD-19 who require ECMO support. Saeed and colleagues set out to evaluate and present the outcomes of these patients.

Methods

The authors report a multicenter, retrospective cohort study evaluating adult patients with COVID-19 who were supported with ECMO. The majority of patients (96%) underwent Veno-venous ECMO (VV-ECMO) cannulation while the remaining 4% were rescued with Veno-arterial ECMO (VA-ECMO). The primary outcome was in-hospital mortality in patients who were placed on ECMO with time to event analysis at 90 days. Additional outcomes were ECMO complications including secondary infections, deep venous thrombosis, stroke, limb ischemia, changes in ECMO configuration or circuit exchange, and renal failure requiring dialysis.

Results

The study evaluated 292 patients who underwent ECMO treatment for COVID-19 infection. Of those patients, 39% expired, 46% were transferred from the hospital alive, 6% remained on ECMO, and 9% were off ECMO but remained hospitalized. Most cases were venovenous ECMO (VV ECMO) and initial cannulation strategy of femoral and jugular veins comprised the majority of patients (47%) while the others underwent bifemoral cannulation (19%) or dual lumen catheter in the internal jugular vein. Patients who survived to discharge or transfer were cannulated 4 days earlier than those patients who expired. In addition, the duration of ECMO was longer in patients who died than those who were discharged. 55% of patients experienced a secondary infection (most commonly bacteremia or pneumonia). Hemorrhagic stroke (6%) and ischemic

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stroke (1%) occurred in 7% of patients. The in-hospital mortality rate at 90 days post ECMO initiation was 42%. The most common causes of death included multi-organ failure, cardiac failure, and respiratory failure. After multivariate analysis, older age, renal dysfunction, and receiving CPR before being placed on ECMO were associated with death during hospitalization. Interestingly, sex, preexisting comorbidities, and length of intubation prior to ECMO initiation were not associated with death. There was geographical variation in hospital mortality, reportedly patients in the Northeast did worse than patients in the Midwest and the South. The Midwest experienced significantly reduced mortality ($p<0.01$).

Discussion

In this large retrospective analysis of patients with COVID-19 placed on ECMO, the authors demonstrated decreased survival after ECMO in older patients, in patients with renal dysfunction, and patients receiving CPR before EMCO cannulation. The reported mortality rates for this US-based cohort were similar to the worldwide Extracorporeal Life Support Organization (ELSO) registry data[4]. The ELSO registry data also identified older age, kidney injury, and pre-ECMO CPR as contributing to mortality; and additionally, they noted immunocompromised states, chronic respiratory disease, and venoarterial ECMO linked with increased mortality. This retrospective data suggests that ECMO may be used judiciously as a treatment option for patients with severe COVID-19 respiratory failure as currently, the morbidity and mortality remain high. ECMO is a scarce resource and ECMO treatment algorithms developed in the future could consider addressing patient age, renal function, and prior CPR in addition to other patient eligibility criteria.

References

1. Karagiannidis C, Brodie D, Strassmann S, Stoelben E, Philipp A, Bein T, Müller T, Windisch W. Extracorporeal membrane oxygenation: evolving epidemiology and mortality. Intensive Care Med. 2016 May;42(5):889-896. doi: 10.1007/s00134-016-4273-z. Epub 2016 Mar 4. PMID: 26942446.
2. Combes A, Peek GJ, Hajage D, Hardy P, Abrams D, Schmidt M, Dechartres A, Elbourne D. ECMO for severe ARDS: systematic review and individual patient data meta-analysis. Intensive Care Med. 2020 Nov;46(11):2048-2057. doi: 10.1007/s00134-020-06248-3. Epub 2020 Oct 6. PMID: 33021684; PMCID: PMC7537368.
3. Ma X, Liang M, Ding M, Liu W, Ma H, Zhou X, Ren H. Extracorporeal Membrane Oxygenation (ECMO) in Critically Ill Patients with Coronavirus Disease 2019 (COVID-19) Pneumonia and Acute Respiratory Distress Syndrome (ARDS). Med Sci Monit. 2020 Aug 6;26:e925364. doi: 10.12659/MSM.925364. PMID: 32759887; PMCID: PMC7430351.
4. Barbaro RP, MacLaren G, Boonstra PS, Iwashyna TJ, Slutsky AS, Fan E, Bartlett RH, Tonna JE, Hyslop R, Fanning JJ, Rycus PT, Hyer SJ, Anders MM, Agerstrand CL, Hryniewicz K, Diaz R, Lorusso R, Combes A, Brodie D; Extracorporeal Life Support Organization. Extracorporeal membrane oxygenation support in COVID-19: an international cohort study of the Extracorporeal Life Support Organization registry. Lancet. 2020 Oct 10;396(10257):1071-1078. doi: 10.1016/S0140-6736(20)32008-0. Epub 2020 Sep 25. Erratum in: Lancet. 2020 Oct 10;396(10257):1070. PMID: 32987008; PMCID: PMC7518880.

Survival Following Alcohol Septal Ablation or Septal Myectomy for Patients with Obstructive Hypertrophic Cardiomyopathy

Hao Cui, Hartzell Schaff, Shuiyun Wang, et. al. J Am Coll Cardiol. 2022 May 3;79(17):1647-165.

Reviewers:
Sohail K. Mahboobi, MD FASA
Lahey Hospital & Medical Center
Burlington, MA

The initial medical management for patients with hypertrophic cardiomyopathy (HCM) and left ventricular outflow tract (LVOT) obstruction is beta-blockers, calcium-channel blocking agents, and/or disopyramide.[1] The surgical management is septal reduction therapy (SRT) by myectomy that can provide near-complete relief of LVOT obstruction with improvement in symptoms and survival.[2] Percutaneous alcohol septal ablation (ASA) is an alternate SRT that reduces septal thickness. The results are similar for both procedures comparing early mortality with septal ablation has higher rates of pacemaker implantation and higher residual LVOT gradient.[3]

Methods

This was a multicenter trial and included review of patients with obstructive HCM who underwent septal myectomy or ASA from 1998 through 2019. Patients with prior septal myectomy or ASA were excluded along with the patients who had a major concomitant cardiovascular procedure such as coronary artery bypass grafting or mitral valve repair for primary valve pathology. The decision for septal myectomy or ASA was made following a full discussion of both procedures with the patient. All-cause mortality was the primary outcome. Differences between treatment groups (ASA vs myectomy) were assessed by using Wilcoxon rank sum tests or Pearson chi-square, respectively.

Results

A total of 3,859 patients were included. The median age was 54.8 years (IQR: 45.8-64.3 years), and 2,115 (54.8%) were male. Patients undergoing ASA were older compared to the septal myectomy (63.0 years [IQR: 52.7-72.8 years] vs 53.7 years [IQR: 44.9- 62.8 years]; P < 0.001). Patients undergoing septal ablation also had more comorbidities. However, the septal thickness was smaller in patients undergoing ASA (19.0 mm [IQR: 17.0-22.0 mm] vs 20.0 mm [IQR: 17.0-23.0 mm]; P ¼ 0.007). There was no difference in preoperative LVOT gradient between the two groups. During the first 30 days following the procedures, there were 4 (0.7%) early deaths in the ASA group and 9 (0.3%) in the myectomy group. Over a median follow-up of 6.4 years (IQR: 3.6-10.2 years), the 10- year all-cause mortality was 26.1% in the ASA group and 8.2% in the myectomy group. The mortality was increased in patients undergoing ASA compared to those undergoing myectomy. After adjustment for age, sex, and comorbidities, the risk of mortality remained greater in patients having septal reduction by ASA (HR: 1.68; 95% CI: 1.29-2.19; P < 0.001). Other variables associated with increased mortality were older age, New York Heart Association (NYHA) functional class III to IV, chronic lung disease, preoperative cerebrovascular accident, atrial fibrillation, renal failure, diabetes, and greater septal thickness. Compared to septal myectomy, ASA had an increased risk of mortality in both men and women. Both groups were well balanced on baseline characteristics by using propensity score.

Discussion

According to the findings of this study, ASA was associated with increased long-term mortality compared to septal myectomy. Septal myectomy provides more immediate and complete relief of LVOT gradients and symptoms compared to ASA, but procedural risks are similar with the 2 methods, and recovery is more rapid with ASA. Some clinicians advise ASA for older patients because of perceived increased operative risk and do not recommend it to young patients because of the risk of heart block and pacemaker dependency. ASA is less effective in relieving LVOT gradients in patients with thick septa (>30 mm).[4]

The occurrence of complete heart block requiring transient and permanent pacing is significantly higher after ASA compared to septal myectomy mostly due to the larger mass of septum affected during ASA.[5] The residual LVOT gradient is higher following ASA compared to septal myectomy and reintervention is necessary in 10% to 20% of patients. The residual gradient may increase long-term mortality. The selection of the method of SRT should be individualized. For patients with limited life expectancy, the benefit of survival may be counterbalanced with potential surgical risk and quality of life. In this study data on postoperative cardiovascular events including implantable cardioverter-defibrillator discharge were not available.

Also, the ultimate effect of ASA in reducing septal thickness and LVOT gradients may not become apparent until 2-3 months and an early evaluation and comparison of gradient relief may not be appropriate.

References

1. Ommen SR, Mital S, Burke MA, et al. 2020 AHA/ACC Guideline for the Diagnosis and Treatment of Patients With Hypertrophic Cardiomyopathy: Executive Summary: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. Circulation. 2020 Dec 22;142(25):e533-e557.
2. Ommen SR, Maron BJ, Olivotto I, et al. Long-term effects of surgical septal myectomy on survival in patients with obstructive hypertrophic cardiomyopathy. J Am Coll Cardiol. 2005 Aug 2;46(3):470-6.
3. Alam M, Dokainish H, Lakkis NM. Hypertrophic obstructive cardiomyopathy-alcohol septal ablation vs. myectomy: a meta-analysis. Eur Heart J. 2009 May;30(9):1080-7.
4. Lu M, Du H, Gao Z, Song L, et al. Predictors of Outcome After Alcohol Septal Ablation for Hypertrophic Obstructive Cardiomyopathy: An Echocardiography and Cardiovascular Magnetic Resonance Imaging Study. Circ Cardiovasc Interv. 2016 Mar;9(3):e002675
5. Valeti US, Nishimura RA, Holmes DR, et al. Comparison of surgical septal myectomy and alcohol septal ablation with cardiac magnetic resonance imaging in patients with hypertrophic obstructive cardiomyopathy. J Am Coll Cardiol. 2007 Jan 23;49(3):350-7

Intraoperative Venous Congestion Rather Than Hypotension is Associated with Acute Adverse Kidney Events After Cardiac Surgery: A Retrospective Cohort Study

Chen L, Hong L, Ma A, Chen Y, Xiao Y, Jiang F, Huang R, Zhang C, Bu X, Ge Y, Zhou J: British Journal of Anaesthesia 2022;128(5):785e795 doi: 10.1016/j.bja.2022.01.032

Reviewer:
Andrew Maslow MD:
Providence Anesthesia,
Rhode Island Hospital, Providence RI

Perioperative renal dysfunction is associated with increased ICU and hospital stays, and greater morbidity and mortality for the cardiac surgical patient. Studies have reported that age, preoperative renal and cardiac function, the cardiac surgical procedure, and surgical duration as predictors of acute kidney injury/dysfunction.[1,2,3,4] Hemodynamic variables such as systemic arterial hypotension and/or cardiogenic shock have also been reported.[2] Varying significance of predictors of perioperative renal dysfunction are, in part, due to variations in patient populations, surgical procedures, and demographics. The role of congestive renal failure in association with elevated venous pressures has been described as a pathophysiologic mechanism of renal dysfunction being described experimentally in the 1930s and periodically revisited thereafter.[5]. **Chen et al** present a relatively simplistic look at hemodynamic variables and their association with acute and prolonged kidney injury in cardiac surgical patients.[6]

Chen et al present the impact of systemic arterial hypotension and central venous congestion on perioperative renal function in 5127 patients undergoing cardiac surgery.[6] The authors hypothesized that duration and magnitude of intraoperative hypotension and venous congestion are associated with acute kidney dysfunction in patients undergoing cardiac surgery. Patients with bypass times < 30 minutes, those undergoing aortic surgery, and patients with prior renal dysfunction were excluded. The authors evaluated the impact of three different systemic mean blood pressure thresholds (mBP < 55, 65, or 75mmHg) or three central venous pressure thresholds (CVP > 12, 16, or 20 mmHg). Intraoperative hypotension and venous congestion were quantified using time spent under each absolute MAP threshold value or above each CVP threshold value, and total area under each MAP threshold time plot or area under the CVP curve above threshold.

Acute Kidney Injury (AKI) as an absolute increase in serum creatinine of 26 mM (mg/dl= 0.0113mM; mM = 88.4mg/dl) within 48 h or an increase in serum creatinine of 1.5 times the baseline value within 7 days. Acute Kidney Disease (AKD) was defined as serum creatinine value elevated to 1.5 times the baseline value between 8 and 90 postoperative days.

1070 (20.9%) and 327 (7.2%) developed acute kidney injury and acute kidney disease respectively. A total of 737 patients developed KDIGO stage 1 AKI, 161 developed stage 2 and 172 developed stage 3 AKI, and the 90 day incidence of RRT was 2.4%. Among patients with AKI, 273 (27.5%) developed AKD. In contrast, 54 (1.5%) patients without AKI developed AKD. Both AKI and AKD were associated with a longer ICU and hospital LOS than patients without acute adverse kidney events.

Overall, 3452 (67.3%), 1714 (33.4%), and 834 (16.3%) patients had episodes with CVP 12, 16, and 20 mm Hg, respectively. Intraoperative hypotension was observed among 4391 (85.6%), 5084 (99.2%), and 5121 (99.9%) patients based on 55, 65, and 75mm Hg MAP.

Acute kidney injury was associated with both hypotension and venous congestion. Acute kidney disease was associated with the duration of any venous congestion. Venous congestion was associated with an 8-17% increased risk of renal replacement therapy while systemic hypotension was not. Venous congestion was associated with AKD while systemic hypotension was not.

Discussion

The authors report the association between intraoperative blood pressure and central venous pressure with renal outcome. Elevated CVP was increasing associated with greater risk for both AKI and AKD while intraoperative reductions in blood pressure was only associated with increased AKI. Disappointingly, the authors did not report how often a high or low blood pressures were associated with a low or high CVPs and whether or not hemodynamic combinations were meaningful. Although these hemodynamic data were associated with renal injury, there is little insight into the cause of blood pressure decreases or CVP elevations. The authors did not provide any on-going assessment of blood flow or heart function. We are left unaware as to whether the patients were experiencing cardiogenic shock, acute pulmonary vascular or right heart issues, or changes in volume status at any given time. While cardiopulmonary bypass management is listed, general hemodynamic management is not described. These additional data would be useful to allow consideration as to how to manage changes in hemodynamic data.

Although details of hemodynamic management and data were not reported nor discussed, it is evident that venous hypertension and presumably congestion is associated renal dysfunction and may be a therapeutic target and/or could be an under-appreciated component of goal-directed-therapy (GDT). The present study identifies a linear relationship between CVP and kidney dysfunction from < 12 to > 20 mmHg.[6] Other data have similarly reported a linear relationship between CVP and renal dysfunction starting with a CVP of 4 mmHg, with several articles reporting that a CVP > 15 was predictive of poor outcome.[2,7,8] In septic patients, an increase of the CVP by 5 mmHg was associated with a 2.7x increase in AKI, which in turn was associated with greater mortality.[8] Few disagree that systemic artery perfusion pressure and cardiac output are important for systemic organ perfusion, however, the value of the CVP varies.[9,10,11]

Across different patient populations, there is little debate regarding variables that affect organ perfusion; 1) flow, 2) perfusion pressure, and 3) pressure gradients, the latter being the difference between afferent arterial pressure and efferent venous pressure. Maintaining a higher perfusion pressure by permissive arterial hypertension and/or by lowing the CVP would be expected to have benefits. Even if it is argued that a CVP of 8, 10, 12, or > 15mmHg may or may not reflect right heart volume, it is important to recognize that organs see flow and pressure and that an elevated venous pressure may adversely affect end organ flow/perfusion.[12] A goal-directed hemodynamic therapy maintaining a CVP between 6-8 mmHg and mBP 65-90 mmHg was reported to be associated with shorter ventilatory time and reduced renal complication for cardiac surgical patients.[13]

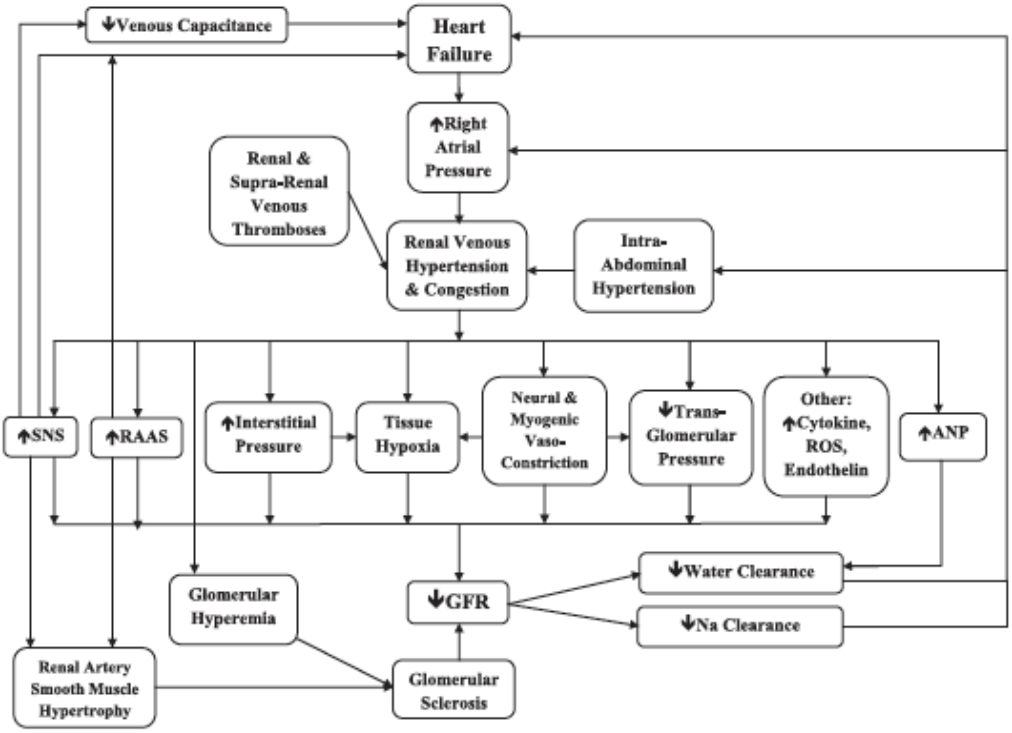
Renal venous hypertension lowers the renal artery-vein pressure gradient and renal blood flow, causes interstitial congestion, and renal dysfunction.[5] When describing hemodynamics of renal dysfunction several pathways are described: 1) heart failure

causing reduced forward flow to the kidneys, 2) heart failure causing increases in right heart, right atrial, and venous pressures, and 3) renal dysfunction causing heart failure (FIGURE 1).[5] As to what the ideal venous pressure should be isn't entirely clear, however, data show a linear relationship between a CVP > 15mmHg and renal dysfunction.[2,3,6]

Recognizing the role of renal venous hypertension and kidney dysfunction allows consideration of therapeutic targets that may be applicable across different patient populations.[5,11] In the sepsis population aggressive hydration may be more harmful especially when coupled with reduced forward flow.[12,14] Combining a lower CVP, permissive arterial hypertension, and a higher CO reduces AKI and associated with improved survival.[11,15] Other therapies to reduce venous congestion include diuretics, sympatholytics and mechanical ultrafiltration each with reported success in improving kidney function.[5]

Confusing or varied conclusions with regard to the value of the CVP highlight issues with different patient populations, varied goal directed therapies, and a misunderstanding by referring to the CVP as a volume monitor only and, perhaps worse, driving up the CVP to aggressively hydrate patients.[16,17,18] Although the elevated CVP in cardiac surgical patients may reflect cardiac dysfunction, a predictor of end organ dysfunction, a relatively simplistic message by Chen et al is that venous pressures affect organ function by affecting organ perfusion.[6]

Given that the same basic principles of end organ perfusion apply to all organs, reducing venous congestion would be expected to have benefits, especially while optimizing end organ arterial perfusion pressure to improve end organ perfusion gradient (systemic arterial pressure - venous pressure).



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