UP CSI NEWSLETTER FEBRUARY 2025

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From the Desk Of The Editor

DR. ROOPALI KHANNA

Dear Readers,

We are delighted to present the latest issue of our newsletter for February 2025. This edition provides an overview of the diverse topics that will be discussed at Cardicon 2025. The authors have shared brief excerpts from their talks, offering readers valuable insights into their presentations.

This issue features a wide range of topics, including hypertension, heart disease in women, heart failure, lipid disorders, coronary artery disease, and cardiometabolic sessions. It also covers the latest advancements in drug management for heart disease and explores the emerging role of artificial intelligence in cardiology. Our contributors have worked tirelessly to bring you relevant and thought-provoking content.

Thank you for your continued support. We hope this issue enriches your knowledge, inspires new ideas, and offers fresh perspectives.

We also look forward to an excellent conference, Cardicon 2025, organized by Dr. Nirdesh Jain, taking place on February 8th and 9th, 2025, in Orchha.

Best Regards,

Dr. Roopali Khanna

Overview Of The Scientific Program

Dear Colleagues

It is my proud privilege to invite you all to 30th Annual Conference of Cardiological Society of India, U.P. chapter at Jhansi. It is one of the most important cardiological meeting of U.P.
The regional and National experts of field of cardiology will be discussing on major clinical areas of cardiology practice.

The Scientific programme we have designed to cover Hypertension, Heart failure, Artificial Intelligence, Lipidology, Coronary artery disease, ECG and Arrythmia, Valvular Heart disease, Congenital Heart disease, Cardio metabolic sessions, Women and heart disease, Cardiovascular pharmacology.

The whole scientific committee and organizing committee hope that this academic feast will be clinically useful, academically stimulating and will provide newer research ideas.

Prof. Roopali Khanna, the editor of our UPCSI news letter has put in tremendous effort to take out this news letter for delegates, UPCSI members and residents, so important excerpts of Academic topics can be put in your hand as ready reference.

Our scientific agenda is also comprising of ECG quiz for residents and physicians, DM quiz for DM residents and programme for CV technician and paramedics on day two, to make this meeting more interesting and exciting.

On behalf of organizing secretary Dr. Nirdesh and entire organizing team I take this apportunity to welcome you all in this scientific feast.

Warm Regard

Dr. A. K. Pandey, MD, DM, FACC, FESC, FSCAI (Cardio).
President Elect - UPCSI and Chairman Scientific Committee
Department of Cardiology
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LIPOPROTEIN (A) AS A THERAPEUTIC TARGET

DR. SATYENDRA TEWARI

SGPGIMS, LUCKNOW

Lp(a) is a plasminogen-like protein produced by the liver and composed of an LDL particle (apo B100) covalently bound to apolipoprotein(a) by a single disulfide bond. Elevated Lp(a) levels are reported in 20-25 % of the world population and it is an independent risk factor for cardiovascular diseases, including coronary artery disease (CAD), stroke, and peripheral artery disease (PAD). High Lp(a) is a common CVD risk factor and conscious efforts, as directed by clinical guidelines and consensus statements, should be made to screen individuals/patients for comprehensive CVD risk profile. Considering the heritable nature of Lp(a) levels, cascade screening of first-degree family members is another warranted. The recent AHA scientific and EAS consensus statements have recommended cascade screening for high Lp(a), including those with a personal or family history.

Targeted apo(a)-lowering therapies that lower plasma Lp(a) levels will enable better management of CVD risk and CVD events. These include lipoprotein apheresis in addition to niacin and PCSK9 inhibitors. Novel therapeutics targeting lp (a) mRNA translation are under trial. Other gene-editing approaches targeting dyslipidemia are on the horizon, offering alternative and exciting opportunities to target atherogenic lipoproteins as a 'one-and-done' therapy. Most notably, CRISPR-based therapeutics have the capacity for genome editing directly at the DNA sequence level and have proven valuable in experimental research settings. Until more targeted therapies are available, patients with high Lp (a) may be treated using the armamentarium of available interventions with a risk-based strategy.

NEW APPROACHES TO REDUCE RECURRENT PCI: TO ANGIOPLASTY AND BEYOND

DR. SHARAD CHANDRA

KGMU, LUCKNOW

INTRODUCTION-

PCI techniques have evolved significantly since its advent in 1977, however approximately 20-40% of patients experience persistent or recurrent angina following PCI. Possible causes include recurrent ischemic lesions from stent thrombosis, in-stent restenosis (ISR), unresolved diffuse disease, myocardial bridging, coronary microvascular dysfunction and inadequate revascularization. The 3 broader strategies to reduce the risk of recurrent PCI can be -

1. Mechanical approach
2. Lipid approach
3. Glucometabolic approach

Mechanical Approach-

Improvements in device technology-

Second and latest generation DES have advanced with improved drug-elution systems and absorbable polymers on the abluminal surface. Thinner struts enhance deliverability and minimize intraluminal obstruction. BIOFLOW V study showed a 40% relative risk reduction in target lesion failure with use of ORSIRO DES, featuring a bioresorbable polymer.

Improvements in revascularization technique with intracoronary physiology-

FFR measurements post stenting have emerged as strong and independent predictors of MACE. A post-PCI FFR of ≥ 0.90 correlates with reduced repeat PCI and MACE. The FFR-SEARCH study found that many patients, despite angiographic success, had suboptimal FFR, with nearly 50% below 0.90 and 10% below 0.80. The DEFINE-PCI study showed that iFR-guided PCI reduced cardiac events, with a post-PCI iFR of ≥0.95 linked to lower rates of TLR.

Improvements in revascularization technique with intracoronary imaging-

IVUS and OCT use has been highlighted in many trials for optimizing PCI and reducing MACE. The IVUS-XPL trial reported IVUS guidance resulted in greater minimum lumen diameter and lower MACE at 12 months, with benefits persisting at five years mainly due to reduction in ischemia driven TLR which align with the observations of ULTIMATE and ADAPT-DES trials. The Pan London study, the largest observational OCT trial, found OCT-guided PCI improved procedural success and reduced in-hospital MACE.Mortality rates were lower with OCT guidance (7.7%) compared to IVUS (12.2%) and angiography (15.7%) across elective and ACS cases.

Lipid approach
Lipid lowering therapies that achieve significant and sustained reduction in LDL-C have shown improved cardiovascular outcomes. Two notable RCTs focused on PCSK9 inhibitors, FOURIER (Evolocumab) and ODYSSEY OUTCOMES (Alirocumab) demonstrated reduction in the composite primary endpoint by 15% individually. Evolocumab reduced repeat coronary revascularizations by 22%, while alirocumab lowered ischemia-driven revascularizations by 12%.
The ORION-4 and VICTORION-2 PREVENT trials are assessing Inclisiran, with results expected in 2025 and 2027. Bempedoic acid reduced LDL-C by 20%–25%, improving coronary outcomes per CLEAR Outcomes. Icosapent ethyl as per JELIS and REDUCE-IT trials also demonstrated reduction in requirement of coronary revascularisation.

Glucometabolic approach- Newer antidiabetic agents go beyond glycemic control and could contribute to reduction of primary or recurrent cardiovascular events. GLP1-RA reduces MACE in diabetic patients, as shown in HARMONY, LEADER, and SUSTAIN 6 although PIONEER-6 was inconclusive. SGLT2i offers cardiovascular protection, with EMPA-REG, CANVAS, DECLARE-TIMI 58, and CREDENCE trials showing a 12% MI risk reduction and 17% lower cardiovascular death. Tirzepatide, a dual GIP/GLP-1 agonist, improves glycemic control and weight loss without increasing hypoglycemia risk per SURPASS trials and can be a promising drug.

Conclusion
These "outside of stent" strategies help improve the risk factors and reduce the residual risk in patients after PCI. We should aim to incorporate optimal medical treatment strategies alongside use of intracoronary imaging and physiology tools for optimal PCI results in order to improve long term outcomes reducing the risk of TLR and recurrent PCI.

MANAGEMENT OF MID-RANGE HEART FAILURE TREATMENT (HFmrEF) BASED ON ACC/AHA GUIDELINES

DR. UMESHWAR PANDEY
LPS, KANPUR

Heart failure with mildly reduced ejection fraction (HFmrEF), defined as a left ventricular ejection fraction (LVEF) of 41-49%, represents an intermediate category between heart failure with reduced ejection fraction (HFrEF) and heart failure with preserved ejection fraction (HFpEF). The American College of Cardiology (ACC) and the American Heart Association (AHA) provide updated guidelines for managing HFmrEF, emphasizing symptom relief, risk factor control, and optimizing guideline-directed medical therapy (GDMT).

Key Management Strategies

1. **Pharmacologic Therapy:**
   - Beta-blockers: Recommended to reduce mortality and hospitalization.
   - Renin-Angiotensin System Inhibitors (ACEi, ARB, ARNI): Angiotensin receptor-neprilysin inhibitors (ARNI) are preferred over ACE inhibitors (ACEi) or angiotensin receptor blockers (ARB) where possible.
   - Mineralocorticoid Receptor Antagonists (MRA): May provide benefit in reducing heart failure progression.
   - Sodium-Glucose Co-Transporter-2 Inhibitors (SGLT2i): Shown to reduce hospitalization and cardiovascular mortality.
2. **Non-Pharmacologic Management:**
   - Lifstyle Modifications: Sodium restriction, weight management, exercise, and smoking cessation.
   - Rehabilitation: Recommended for stable patients to improve functional capacity and quality of life.
3. **Device Therapy & Other Interventions:**
   - Implantable Cardioverter-Defibrillators (ICD)
   - Considered in patients with arrhythmias or worsening LVEF.
   - Cardiac Resynchronization Therapy (CRT): May be used in selected patients with conduction abnormalities.
4. **Management of Comorbidities:**
   - Control of hypertension, diabetes, atrial fibrillation, and coronary artery disease is crucial.

Conclusion:
The ACC/AHA guidelines highlight a personalized approach to HFmrEF treatment, incorporating evidence-based pharmacotherapy, lifestyle changes, and careful monitoring of comorbid conditions. Given the evolving research in HFmrEF, ongoing reassessment of patients is essential to optimize treatment outcomes.

CARDIOVASCULAR DISEASE IN WOMEN: HOW IMPORTANT ARE THE BIOMARKERS- LIPOPROTEIN(A), HIGH SENSITIVITY C REACTIVE PROTEIN, LOW DENSITY LIPOPROTEIN CHOLESTEROL

DR. MONIKA BHANDARI

KGMU, LUCKNOW

Established cardiovascular (CV) risk factors such as smoking, diabetes, hypertension, & elevated total cholesterol (TC), & risk prediction models based on such factors, perform well but do not perfectly predict future risk of CVD. Blood biomarkers can be instrumental for understanding biologic processes & for targeting CV interventions. Biomarkers can help identify women at risk for CV disease (CVD) & monitor their condition. They also help researchers understand how CVD affects women differently than men. Thus, there has been much recent interest among cardiovascular researchers in identifying novel biomarkers to aid in risk prediction. In 2007, the Reynolds Risk Score models A (RRS-A) and B (RRS-B) were developed to more accurately identify women's risk of coronary revascularization, myocardial infarction (MI), coronary heart disease death, stroke, or stroke death. By adding high sensitivity C reactive protein (hs-CRP), Lipoprotein(a) [Lp(a)], and family history of CAD, RRS reclassified 40% to 50% of women at intermediate risk based on the National Cholesterol Education Program Adult Treatment Panel III guidelines into higher or lower risk categories.[1] Copenhegan General population study involving 37,545 females and 32,497 males has found that Lp(a) fluctuates in females from menarche to menopause and selectively rises above 50 years of age. They also found that Lp(a) levels more than 40mg/dl predicted similar risk of morbidity and mortality in both sexes above 50 years of age implying that it should be considered an important risk factor for CVD in females above 50 years.[2]

Study done by Manson et. Al, postulated that risk prediction models for CVD that rely on traditional CV risk factors are useful for estimating a woman's risk for developing CVD, but there is room for improvement. They found that alternative lipids, B-type natriuretic peptides, high-sensitivity troponin, CAC, & genetic markers have been could be the novel risk factors that may improve risk prediction. [3]

One study hypothesized that measuring LDL-C, hs-CRP & Lp(a) at single point of time might be useful to assess lifetime risk. They measured Hs-CRP, LDL-C, and Lp(a) levels measured at baseline in 27,939 from women health study of 1992 & were subsequently followed for 30 years. The primary end point was a first major adverse cardiovascular event, which was a composite of myocardial infarction, coronary revascularization, stroke, or death from cardiovascular causes. The study population was then stratified according to quintiles for each biomarker, with quintile 1 including the lowest biomarker levels and quintile 5 the highest levels. Levels of hs-CRP, LDL-C, and Lp(a) were found to have an independent contributions to risk, and the greatest risk was estimated from models that used all three biomarkers in combination. [4]

Conclusions:

- CV health should be assessed routinely in women from a young age.
- This should involve review of traditional CV risk-enhancing factors, sex-specific CV risk factors, and other novel risk factors or biomarkers.
- Biomarkers of inflammation like hs-CRP and markers such as Lp(a), CAC are independent risk predictor as well as add to risk estimation in females especially in intermediate risk category apart from LDL -C
- Sex differences in the predictive role of novel cardiovascular biomarkers for primary prevention require additional study, as do the diagnostic and prognostic utility
- Consideration of other exposures that are unique to or more prevalent in females, such as history of pregnancy complications or autoimmune disease, may also help improve accuracy of risk estimates in women.

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HEART FAILURE WITH PRESERVED EJECTION FRACTION (HFpEF) AND ATRIAL FIBRILLATION (AF): A DUAL CHALLENGE

DR. ADITYA KAPOOR

SGPGIMS, LUCKNOW

Introduction

Atrial fibrillation (AF) is common in patients with heart failure with preserved ejection fraction (HFpEF) and both frequently coexist. Management needs a multi-disciplinary approach.

Lifestyle and General Management for AF in HFpEF

The RACE 3 trial showed that treating comorbid conditions such as hypertension, hyperlipidemia, obesity, heart failure leads to better maintenance of sinus rhythm in persistent AF patients. The trial randomized 245 patients with early persistent AF and mild-to-moderate heart failure (HF) to conventional therapy (n=126) vs targeted therapy (n=119), with the primary endpoint being sinus rhythm at 1 year (with 7 days of Holter monitoring). **Cardiac rehabilitative therapy (physical activity, dietary restrictions, and counselling) was associated with better rhythm maintenance at 1 year** (OR 1.765, , P = 0.042). It was shown in the **LEGACY trial** that a **10 % reduction in weight leads to a 6-fold reduction of probability of AF recurrence**, resting and exercise hemodynamics, epicardial fat, and risk of incident HFpEF. Exercise training in HFpEF patients lead to improved cardiorespiratory fitness and quality of life. The role of continuous positive airway pressure therapy in AF- HFpEF and sleep apnea patients is of unproven value

Medical Therapy for HFpEF With AF

SGLT2 inhibitors – The PRESERVED-HF and EMPEROR-Preserved trials showed that the benefit of SGLT2 inhibitors in HFpEF had no interaction by AF status. Trials with SGLT2i in HFpEF show that the composite endpoint of cardiovascular death or first hospitalization for HF, was consistent for those with AF (HR 0.77, 95% CI 0.69–0.87) and those without (HR 0.83, 95% CI 0.72–0.95)

Direct oral anticoagulants- Patients are at high risk of embolic events due to AF and LA myopathy and such patients should be given oral anticoagulation for stroke prevention.

LAAO devices- Left atrial appendage occlusion (LAAO) devices have been reported to be non-inferior for stroke prevention in these patients.

Rhythm vs rate Control Strategies in HFpEF

Although there is no guideline directed specific therapy for HFpEF-AF patients, the AFFIRM trial showed better results in HFpEF-AF patients who were able to maintain sinus rhythm. Beta blockers and non-dihydropyridine calcium channel blockers are recommended as first line agents for adequate rate control in HFpEF-AF patients. The RATE-AF trial, showed similar efficacy of bisoprolol vs digoxin in elderly HFpEF-AF patients with better secondary outcome parameters such as functional ability and decrease in NT-proBNP in digoxin arm at 12 months.

Anti arrhythmic medications vs AF ablation- In the EAST-AFNET4 trial, it was reported that early rhythm-control therapy was associated with a lower risk of adverse cardiovascular outcomes than usual care among patients with recent AF (diagnosed within 1 year) and underlying cardiovascular conditions (HF n = 798, most of whom had HFpEF). The highest improvement in NYHA class occurred in patients with HFpEF. The exploratory analyses of AF patients in the EAST-AFNET 4 study suggested that **amiodarone, but not treatment with flecainide, propafenone, or dronedarone, was potentially associated with early HF hospitalisations in patients with HFpEF,** suggesting that further clinical research is needed to define the optimal antiarrhythmic drug therapy in patients with HFpEF.

A post-hoc analysis of the **CABANA clinical trial** (ablation vs anti arrhythmic drugs) in HFpEF-AF patients (79% patients in the trial had HFpEF), showed that catheter ablation was associated with 36% relative reduction in the primary composite endpoint.

AV node ablation and permanent pacemaker implantation- may be beneficial in patients of HFpEF with failed rate and rhythm control.

The ABC pathway (Atrial Fibrillation Better Care) pathway):

A. Avoidance of stroke with the use of anticoagulation
B. Better management of symptoms with symptom-directed patient centric decisions on rate or rhythm control
C. Cardiovascular and coexisting-condition risk management, including attention to psychological factors and lifestyle Adherence to the ABC pathway can be associated with better outcomes in patients with AF and HFpEF.

THE ROLE OF AI IN MANAGEMENT OF HYPERTENSION

DR. ASIF HASAN

AMU, ALIGARH

Hypertension is a major modifiable risk factor for coronary artery disease (CAD), which continues to be a major cause of morbidity and mortality globally. Driven by data-centric algorithms, artificial intelligence (AI) has become a game-changing tool for controlling and comprehending CAD and the risk considerations that go along with it. Applications of AI in the management of hypertension include greater prognostication, better risk classification, customized medication, and accurate blood pressure (BP) monitoring via wearable technology. Superior diagnostic and predictive abilities have been shown by machine learning (ML) and deep learning (DL) models, especially when it comes to detecting secondary hypertension and masked uncontrolled hypertension using clinical markers like office blood pressure, pulse pressure, and beta-blocker use, as well as electronic health records (EHRs).

A known independent cardiovascular risk factor is blood pressure variability (BPV). AI-based clustering approaches have classified individuals according to their BPV levels, providing new information about risk assessment. Additionally, when assessing the 10-year risk of cardiovascular events, AI-driven prediction models have performed better than traditional risk calculators like the ACC/AHA CVD risk calculator and the Framingham score. AI is also essential to digital health interventions, enabling lifestyle changes, encouraging self-monitoring, and raising patient awareness.

Notwithstanding these developments, thorough validation and cooperation with medical experts are still necessary for the clinical usefulness of AI in CAD therapy. To guarantee accuracy, dependability, and patient-centered treatment, interdisciplinary efforts are required when integrating AI-driven models into clinical practice. AI has a lot of promise for improving CAD prognosis, prevention, and treatment as it develops further.

BALANCING THE TIGHT ROPE-THERAPY INITIATION AND CONTINUATION IN HF & CKD PATIENTS.

DR. AWADHESH SHARMA

LPS, KANPUR

ABSTRACT:CKD plays a crucial role in the pathophysiology and prognosis of HFrEF and is often a perceived limitation in the optimization of evidence-based HFrEF therapies. With more severe CKD stages, prognosis worsens, and scientific evidence becomes scarce.

Available evidence suggest that most guideline directed medical therapies are effective up to CKD stage 3B, and some drug classes have even shown efficacy in CKD stage 4. There is more evidence for CKD stage 1 to 4 for preventing cardiovascular death/HF hospitalization with evidence-based treatments compared with preventing all-cause mortality. Among treatments, there is some evidence for efficacy of SGLT2i, omecamtiv-mecarbil, ACEi, digoxin, and vericiguat in CKD stage 4. Overall the renal safety profile in all classes of CKD with essentially all treatments is good if the clinical status is taken into account and renal function and potassium are checked regularly.

Many therapies influence renal function directly or indirectly, as well as associated conditions such as hyperkalemia, warranting close monitoring during initiation. A decrease in eGFR (upto 30% of baseleine)is expected with initiation of RAASi (including ARNI) and SGLT2i and should not be a reason to discontinue these life-saving drugs. Renin angiotensin system inhibitors (and probably SGLT2i) cause efferent vasodilation, leading to higher RBF, lower GFR, and lower FF. It is postulated that SGLT2i has effects on afferent arteriolar tone, causing lower RBF, lower GFR, and stable FF. ARNIs may vasodilate the afferent arteriole, causing slightly increased RBF and possibly more preserved GFR (compared with ACEi/ARB alone). ARNIs also influence podocyte function, which may be a factor in the modest albuminuria associated with these drugs.

Knowledge, correct interpretation, and possible treatment of changes in renal function in relation to evidence- based HFrEF treatments are therefore essential assets for optimal HF management.

RENAL DENERVATION

DR. SUBHASH CHANDRA
BLK MAX, DELHI

Renal denervation (RDN) is a minimally invasive, catheter-based procedure aimed at treating resistant hypertension and other cardiovascular conditions. The technique involves disrupting the sympathetic nerve activity along the renal arteries, which play a significant role in blood pressure regulation. This article explores the background, mechanisms, clinical applications, and current advancements in renal denervation.

Background

Hypertension, commonly known as high blood pressure, is a major risk factor for cardiovascular diseases, including stroke, heart failure, and chronic kidney disease. While lifestyle modifications and pharmacological interventions effectively manage hypertension in most cases, some patients exhibit resistant hypertension—a condition where blood pressure remains uncontrolled despite the use of three or more antihypertensive medications, including a diuretic.

Resistant hypertension is partly attributed to heightened sympathetic nervous system activity, which contributes to vasoconstriction, increased cardiac output, and sodium retention. Renal denervation was developed as a targeted approach to modulate this sympathetic activity and provide a treatment option for these patients.

Mechanism of Action

The renal sympathetic nerves, located within the adventitia of the renal arteries, influence kidney function and systemic blood pressure through several pathways:

- **Afferent Pathway**: Transmits signals from the kidneys to the central nervous system, stimulating sympathetic outflow.
- **Efferent Pathway**: Sends signals from the central nervous system to the kidneys, promoting sodium retention, renin release, and vasoconstriction.

RDN involves the use of a catheter equipped with radiofrequency energy, ultrasound, or chemical ablation to disrupt these nerves. The catheter is inserted into the renal artery via the femoral artery, and the energy delivery selectively ablates the sympathetic nerve fibers, reducing their activity and ultimately lowering blood pressure.

Clinical Applications

1. **Resistant Hypertension:** The primary application of RDN is in patients with resistant hypertension. Multiple studies have demonstrated significant and sustained reductions in blood pressure following the procedure.
2. **Heart Failure:** Emerging evidence suggests that RDN may improve outcomes in patients with heart failure by reducing sympathetic overactivity.
3. **Chronic Kidney Disease (CKD):** RDN has shown potential in reducing renal inflammation and improving renal function in certain CKD populations.
4. **Other Conditions:** Research is ongoing to explore the role of RDN in treating arrhythmias, diabetes mellitus, and metabolic syndrome.

Clinical Evidence

Several landmark trials have evaluated the safety and efficacy of RDN:

- **SYMPLICITY HTN-2:** Demonstrated significant blood pressure reductions in patients undergoing RDN compared to a control group.
- **SPYRAL HTN-OFF MED and SPYRAL HTN-ON MED:** Highlighted the efficacy of RDN in both medicated and unmedicated patients with hypertension.
- **RADIANCE-HTN:** Showed promising results using ultrasound-based RDN technology.

While earlier studies raised concerns about inconsistent results, advancements in technology and better patient selection criteria have improved outcomes in recent trials.

Advantages and Risks

Advantages:

- Minimally invasive procedure.
- Durable blood pressure reduction.
- Potential to reduce the reliance on antihypertensive medications.

Risks:

- Vascular complications at the access site.
- Potential for renal artery stenosis.
- Uncertain long-term outcomes in some populations.

Current Trends and Future Directions

The field of renal denervation continues to evolve with innovations such as:

- **Improved Ablation Techniques:** Development of multi-electrode catheters for more efficient nerve disruption.
- **Expanded Indications:** Exploration of RDN for broader applications, including atrial fibrillation and obesity.
- **Combination Therapies:** Integration of RDN with pharmacological approaches for synergistic effects.

Ongoing research and long-term studies will further clarify the role of RDN in clinical practice and help refine patient selection criteria to maximize its benefits.

Conclusion

Renal denervation represents a promising therapeutic option for resistant hypertension and other cardiovascular conditions. With advancements in technology and growing clinical evidence, it has the potential to become a cornerstone treatment for managing sympathetic overactivity. However, careful patient selection, thorough understanding of the procedure, and continued research are essential to fully realize its potential.

SEQUENTIAL CHAMBER ANALYSIS BY ECHOCARDIOGRAPHY

DR. NEERAJ AWASTHY

FORTIS OKHLA, DELHI

Echocardiographic evaluation of congenital heart lesions relies on the principles of sequential chamber analysis, whereby the heart is analyzed in a segmental fashion in terms of its atrial, ventricular, and arterial components.

PRINCIPLES OF SEQUENTIAL CHAMBER ANALYSIS:

The cardinal principle of sequential chamber analysis states that the morphology of a chamber should be determined based on its most constant component.

Steps in Sequential Chamber Analysis:

1. Identification of Atria and Atrial Arrangement
2. Identification of Ventricular Morphology and Atrioventricular Connection
3. Identification of Great Vessels and Ventricular-Arterial Connection
4. Identification of Associated Abnormalities and Assessing Their Severity

IDENTIFICATION OF ATRIA AND THEIR ARRANGEMENT:

The first step in sequential analysis is identifying atrial arrangement, which can be classified into:

1. Normal atrial arrangement (Situs solitus): The right atrium is right-sided, and the left atrium is left-sided.
2. Mirror image atrial arrangement (Situs inversus): The right atrium is left-sided, and the left atrium is right-sided.
3. Isomerism of the left atrial appendage: Both atrial appendages resemble the left atrial appendage.
4. Isomerism of the right atrial appendage: Both atrial appendages resemble the right atrial appendage.

Atrial Identification Markers:

a. Atrial Appendage:
   - The right atrial appendage is broad and triangular, with coarse pectinate muscles extending around the atrioventricular junction.
   - The left atrial appendage is tubular with a narrow junction and has smooth posterior walls.

b. Venous Component:
   - The opening of the suprahepatic portion of the inferior vena cava (IVC) into the right atrium is a key marker.

c. Atrial Septum:
   - The septum primum overlaps the secundum from the left atrial side.

ECHOCARDIOGRAPHIC IDENTIFICATION OF ISOMERISM:

Right isomerism (asplenia syndrome) and left isomerism (polysplenia syndrome) can be suspected based on the arrangement of abdominal great vessels.

Right Isomerism:

- Aorta and IVC lie on the same side of the spine.
- The IVC may open into either atrium or the midline.

Left Isomerism:

1. Aorta and a venous channel lie on the same side of the spine.
2. The venous channel represents the azygos/hemiazygos system.
3. IVC interruption is common, with hepatic veins draining directly into the atrium.

ATRIOVENTRICULAR CONNECTIONS:

Atrioventricular connections describe how atrial myocardium connects to the ventricular mass. The types include:

1. Concordant Atrioventricular Connection
2. Discordant Atrioventricular Connection
3. Univentricular Atrioventricular Connection
4. Isomeric Atrioventricular Connection
5. Uniatrial and Biventricular Connection

Echocardiographic Identification of Ventricular Morphology:

1. Offsetting Sign of Atrioventricular Valve:
   - The tricuspid valve is more apically positioned than the mitral valve.
2. Ventricular Apical Trabeculation:
   - Right ventricle has coarse trabeculations with a prominent moderator band.
3. Papillary Muscle Arrangement:

◦ The left ventricle has two discrete papillary muscles.

VENTRICULOARTERIAL CONNECTIONS:

1. Concordant Ventriculoarterial Connection:
   - The left ventricle connects to the aorta, and the right ventricle connects to the pulmonary artery.
   - Discordant Ventriculoarterial Connection:
   - The left ventricle connects to the pulmonary artery, and the right ventricle connects to the aorta (transposition of great arteries)
2. Double Outlet Ventricle (Right or Left):
   - Both great vessels arise predominantly from one ventricle.
3. Single Outlet (Pulmonary or Aortic Atresia):

Only one great vessel is patent. Echocardiographic Approach to Identify Ventricular-Arterial Connection: a) Pulmonary Artery Identification:

- The pulmonary artery bifurcates early and courses posteriorly.
- The aorta courses anteriorly and does not branch before the carotids. b) Subxiphoid and Apical Views:
- Gradual superior tilting of the transducer helps trace the great vessels' origin.

ATRIOVENTRICULAR CONNECTIONS AND THE LOOP RULE:

D-loop (Right-hand topology): The right ventricle is to the right of the left ventricle in situs solitus and vice versa in situs inversus. L-loop (Left-hand topology): The right ventricle is to the left of the left ventricle in situs solitus and vice versa in situs inversus.

CONCLUSION:

Echocardiography is an indispensable tool in the evaluation of congenital heart disease using the sequential chamber analysis approach. It enables precise identification of atrial, ventricular, and arterial components, allowing for comprehensive diagnosis and management of congenital cardiac malformations.

INOCA – NOT A BENIGN ENTITY

DR. ROOPALI KHANNA

SGPGIMS, LUCKNOW

Ischaemia with Non-Obstructive Coronary Arteries (INOCA) is a prevalent yet underrecognized condition affecting a significant proportion of patients with angina and myocardial ischaemia. More common in women, INOCA is often linked to coronary microvascular dysfunction (CMD) and vasomotor abnormalities, which contribute to myocardial ischaemia despite the absence of obstructive coronary artery disease (CAD). This condition is associated with a high burden of symptoms, recurrent hospitalizations, and an impaired quality of life, comparable to obstructive CAD.

The underlying causes of INOCA are not fully understood, but cardiometabolic risk factors, endothelial dysfunction, oxidative stress, and inflammation contribute to microvascular injury and CMD. Additionally, cardiac autonomic dysfunction has been linked to abnormal vasoreactivity and persistent symptoms.

Despite its impact, INOCA is frequently misdiagnosed or overlooked, leading to inadequate treatment. Advanced non-invasive imaging techniques (TTDE, MCE, PET, MRI, SPECT) and invasive coronary function testing can aid in accurate diagnosis by differentiating between vasospastic angina, microvascular angina, and non-cardiac pain.

Management of INOCA requires a tailored, stratified approach, incorporating lifestyle interventions, cardiovascular risk reduction, and pharmacotherapy to alleviate ischaemia and symptoms. Emerging therapies, including calcium channel blockers, beta-blockers, nicorandil, ranolazine, offer symptom relief for specific INOCA subtypes.

Given the limited mechanistic understanding of INOCA, further research is essential to improve diagnostics and develop innovative, personalized treatment strategies to enhance patient outcomes and quality of life.

CAN BODY ROUNDNESS INDEX (BRI) REPLACE BODY MASS INDEX (BMI)?

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KGMU, LUCKNOW

Introduction: Obesity is a global epidemic with increasing prevalence in children and adolescents. It is one of the top five risk factors for mortality world-wide. The American Heart Association (AHA) has reclassified obesity as a 'major, modifiable risk factor' for coronary heart disease (CHD). Obesity has been shown in several studies to be associated with increasing risk of major cardiovascular risk factors and increasing cardiovascular mortality. Various anthropometric indices have been developed to measure obesity.

Traditionally, Body Mass Index (BMI) has been used as a parameter to quantify obesity, with values below 18.5 depicting malnutrition, 18.5-24.9 being the normal range, 25-29.9 overweight and 30 obese. However, it does not distinguish between fat mass and fat-free mass. So, a more muscular lean person may have similar BMI as an obese fatty person as muscles are denser than fat. Also, BMI does not reflect on the distribution of fat in the body. Individual with higher amounts of fat mass have a larger body volume and thus a larger waist size. Indians are genetically predisposed to gain more weight around the abdomen, and it is well known that visceral fat deposits leading to increasing abdominal circumference are associated with diabetes and worse cardiovascular outcomes. Dr Thomas, a mathematician devised Body Roundness Index (BRI) in 2013 which is a more direct measure of visceral fat1. Based on the concept of eccentricity of planetary orbits given by Johannes Kepler, it tells about the roundness of the body. The values range between 1-16, with 1 being leaner and 16 being more circular. Along with waist circumference and height, BRI calculator uses hip circumference and weight to give information about the % body fat and % visceral adipose tissue (figure 1).

Current evidence: Increasing prevalence of childhood obesity in recent years is paving way for increased incidence of dyslipidemia, diabetes and cardiovascular diseases in early adulthood.

A study published in JAMA on a cohort of more than 30,000 adults showed a trend for increasing BRI over 20 year-period2. There was a U-shaped relation with all cause mortality.

Individuals with a BRI of less than 3.4 had a 25% increased conducted

mortality risk compared to the healthy range, while those with a BRI of 6.9 had a 49% increased risk. Another analysis

of almost 10,000 adults in China older than age 45, from 2011 to 2020, determined that having a higher BRI level over a 6-year period was associated with an increased risk of cardiovascular disease by as much as 163%, even when medical, lifestyle and demographic factors were not considered3. Higher quartiles of BRI were associated with greater all-cause and cardiovascular mortality4 (figure 2).

Conclusion: More recent anthropometric indices measuring visceral fat like BRI result in better cardiovascular risk stratification. They better correlate with metabolic syndrome, cardiovascular and all-cause mortality. BRI can be used as an alternate obesity measurement tool over BMI to better quantify and classify body fat distribution. However, its use requires a calculator or an online tool owing to its complex formula.

Suggested reading:

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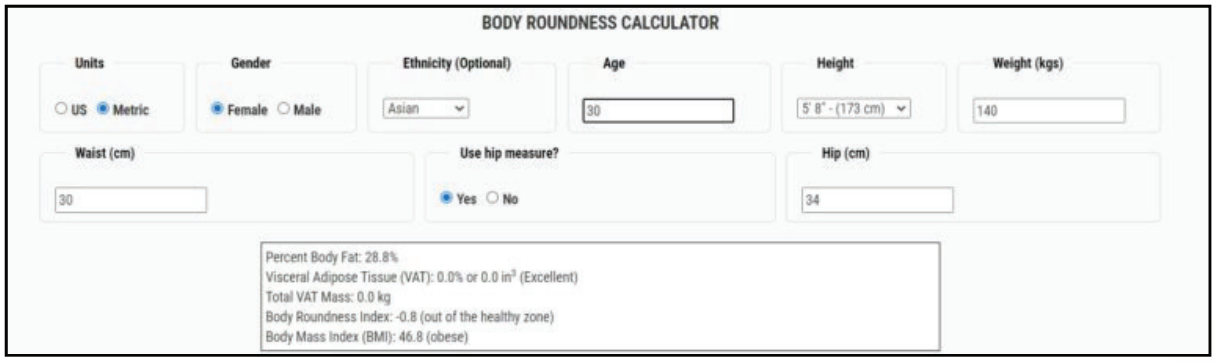
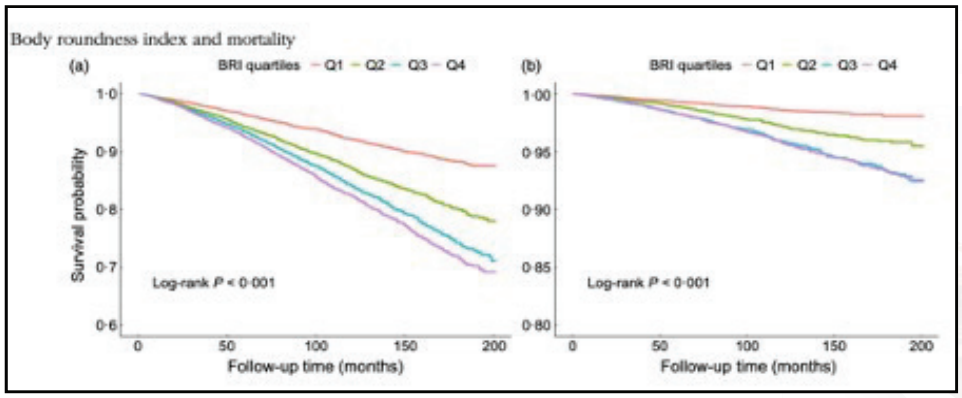
Figure legends:

Figure 1. Screenshot of online Body Roundness Index (BRI) calculator.

Figure 2. Kaplan–Meier survival curve for all-cause(a) and cardiovascular (b) mortality by BRI quartiles.





Beyond Thrombolytic Therapy: What is new in Cardiologist's Hand for Thrombus- The AVIS Protocol

DR. TANUJ BHATIA

SMIH, DEHRADUN

The battle of PCI and thrombolysis for management of acute STEMI, that lasted a few decades, eventually made Primary PCI emerge as a winner, but thrombolysis continued to serve as a backup plan in certain subsets as well as a part of the pharmacoinvasive strategy where Primary PCI would not be offered. But our thirst of better pharmacotherapy still persists: evidence lays in the fact that we still wish to get rid of the large thrombus burden by other means rather than just putting a metal adjacent to that large thrombus inside the coronary vessel. And hence, our focus should move away from the century old, though evidence based but venerable technology of ST elevation to combining our knowledge of pathophysiology and mechanisms of acute myocardial infarction – a "mechanistic" approach to ACS.

Large thrombus burden (LTB) in ACS as per TIMI thrombus grading increases the likelihood of PCI MACE rates, stent thrombosis and no reflow, consequently leading to lower survival rates, threefold increase in mortality and HF related hospitalizations in those who have microvascular obstruction (MVO), and though it always appeared intuitive that thrombus aspiration shall benefit Primary PCI, manual thromboaspiration methods disappointed us in three major RCTs – TOTAL, TASTE and TAPAS and their metanalysis, by not only not being capable of reducing mortality but also being associated with transient ischemia and strokes. They also require syringe changes between procedures, resulting in suboptimal thromboaspiration. This can increase the risk of microvascular obstruction or distal embolization. Hence, 2021 ACC/AHA and 2023 ESC guidelines labelled thromboaspiration as COR III : LOE A, putting a transient stop to our "unmet aspirations".

The CHEETAH study however redefines this strategy with use of sustained mechanical aspiration thrombectomy, in contrast to the manual thrombus aspiration system earlier, with distal embolization rates as low as 0.75%, 96.5% freedom from MACE at 30 days, "zero" device related SAEs, successful thrombus removal with 85% TIMI 2-3 flow post Cat Rx and 97.5% TIMI 3 flow at final angiogram and MBG 3 in 99.8% patients.

The Indigo™ CAT™ RX aspiration system provides up to -29 Hg continuous vacuum pressure using the Penumbra ENGINE® aspiration pump and is compatible with a 0.014-inch guidewire and 6F guide catheter. The consistent suction ability and excellent trackability enabled by its hypotube technology with an atraumatic tip that can navigate seamlessly even through tortuous vessels.

Our experience highlights the importance of following the sequence of Aspiration, Vasodilators, Imaging, and Stenting—the AVIS protocol by an approach that we call as "Minimalism to Maximalism". With this strategy, there was lesser use of pre-dilatation balloons, overall lesser stent lengths with lower vessel coverage at the sites with healthy intima on imaging, better intra-procedural imaging with lesser thrombus burden and microvascular obstruction, and lower chances of no-reflow, distal embolization, or stroke; with higher post-dilatation balloon pressures, larger stent diameters, and more endogenous or exogenous vasodilators reaching the distal bed and therefore, ensured improved MBG, and enabled both multivessel PCI and bifurcation PCI in the same sitting. However, larger multicenter studies employing the AVIS protocol need to validate these findings.

In the present study, patients with ostial LAD with dominant LCx, left main coronary artery (LMCA), ectatic vessels, and slow flow after balloon angioplasty may have benefitted the most from thromboaspiration.

Further, the AVIS protocol may have the potential to reduce major adverse cardiovascular events (MACE), including stroke, improve MBG, navigate tortuous vessels, lower aspiration time, enable better visualization, and improve thrombus debulking and ejection fraction, lead to better understanding of the pathophysiology of ACS and develop novel protocols for metal less PCI, in certain subsets and an excellent alternative option for the high bleeding risk (HBR) population.

PITFALLS IN BP MEASUREMENT

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Patient-related factors: Acute meal ingestion, caffeine or nicotine use, full bladder, and white-coat effect.

Procedure-related factors: Incorrect cuff size, cuff positioning, patient positioning, and talking during measurement.

Equipment-related factors: Using unvalidated devices and incorrect cuff inflation.

Solutions

Standardize measurement techniques: Use a standardized approach to BP measurement.

Use automated devices: Automated devices can reduce errors and improve accuracy.

Provide ongoing training: Educate healthcare professionals on proper BP measurement techniques.

Monitor BP regularly: Regular BP monitoring can help identify errors and improve patient outcomes.

Address patient-related factors: Ensure patients are prepared for measurement, avoiding factors that can affect readings.

Validate devices: Only use devices that have been clinically validated for accuracy.

Benefits of HBPM and ABPM:

Improved accuracy: Both HBPM and ABPM provide more accurate blood pressure readings than office measurements.

HBPM:

Reduces white-coat effect: HBPM helps to eliminate the anxiety-related increase in blood pressure seen in clinical settings:

Provides multiple readings: HBPM allows for multiple readings to be taken over time, providing a more accurate representation of blood pressure.

Improves patient engagement: HBPM empowers patients to take an active role in monitoring their blood pressure, promoting better self-care.

Enhances diagnosis: HBPM can help identify masked hypertension, where blood pressure is normal in the clinic but elevated at home.

ABPM:

Provides 24-hour monitoring: ABPM tracks blood pressure over a 24-hour period, providing a comprehensive picture of blood pressure patterns.

Detects nocturnal hypertension: ABPM can identify nocturnal hypertension, which is associated with increased cardiovascular risk.

Identifies blood pressure variability: ABPM helps to assess blood pressure variability, which is a predictor of cardiovascular events.

Enhances diagnosis and treatment: ABPM can aid in the diagnosis of resistant hypertension, white-coat hypertension, and masked hypertension, allowing for more targeted treatment.

GENE THERAPY IN HEART FAILURE

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Cardiovascular disease is the leading cause of death worldwide. Despite significant advancements in promoting cardiovascular health with therapeutic agents and devices as well as an improved understanding of the molecular pathways underlying cardiovascular disease, the prevalence of heart diseases is increasing. While progress in conventional treatment modalities like drug, device, and cardiac surgeries is making steady and incremental gains to reduce the burden of cardiovascular disease, an urgent need to explore new therapeutic approaches remains. Gene therapy has recently shown promise in the treatment of cardiovascular diseases. Recent advancements illuminate the significant role of genetics in cardiovascular disease etiopathogenesis, encompassing both polygenic and monogenic mechanisms and identifying appropriate targets for gene therapies. Innovative strategies have been developed to tackle pathogenic variants that cause monogenic disorders such as hypertrophic, dilated, and arrhythmogenic cardiomyopathies and hypercholesterolemia. These include the delivery of indexed genes to supplement insufficient

protein levels caused by pathogenic variants or genome editing to correct, delete, or modify mutant sequences to correct protein function. However, effective delivery of indexed genes to specified cells like the heart always poses formidable challenges. Viral vectors, notably adeno-associated viruses and nonviral vectors such as lipid and engineered nanoparticles, offer distinct advantages and inherent limitationsAdeno-associated viral vector-based gene delivery and CRISPR-Cas9-based genome editing have emerged as efficient strategies for gene delivery in humans.. Strategies to modulate gene expression using antisense oligonucleotides or small interfering RNA are proving to be safe and effective in the clinic. Clinical success has been reached primarily with liver-directed therapies that target genes of risk factors underlying heart failure or diseases in which proteins produced by the liver affect the heart and its vasculature. However, gene therapies targeting the cardiac muscle have not been successful so far. Overall, gene therapy holds the promise of expanding current treatment options and intervening in previously untreated genetic heart disease mechanisms with few side effects.

ELEVATED LDL CHOLESTEROL

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Elevated LDL cholesterol is one of the primary initiating factors for atherosclerotic cardiovascular disease (ASCVD) and is considered the most critical, yet modifiable, risk factor among the top three for cardiovascular disease. Long-term exposure to high LDL-C is a significant predictor of ASCVD, as evidenced by over 200 studies involving more than two million patients, 20 million person-years of follow-up, and more than 150,000 cardiovascular events. These studies, encompassing genetic research, cohort analyses, and randomized trials, have shown a dose-dependent, log-linear relationship between LDL-C levels and the risk of acute cardiovascular events. Clinical trials consistently demonstrate that lowering LDL-C reduces the risk of major cardiovascular events, showing a 22% relative risk reduction per 1 mmol/L decrease in LDL-C.

Real-world evidence indicates a substantial unmet need in achieving LDL-C targets, with over 80% of patients post-acute coronary syndrome (ACS) failing to reach guideline-recommended LDL-C levels despite statin therapy. Only 30% of patients achieve an LDL-C level of less than 55 mg/dL, and 45% reach less than 70 mg/dL. The Cardiological Society of India (CSI) 2023 guidelines categorize patients into various risk categories, recommending LDL-C goals of less than 55 mg/dL for very high-risk patients and less than 85 mg/dL for non-HDL-C. Aggressive lipid-lowering strategies are universally supported by guidelines, emphasizing 'the lower, the better' approach in managing lipid levels, especially in ACS patients.

For lipid-lowering treatment, a combination therapy approach is often required to achieve optimal LDL-C goals. While traditional therapies such as statins, ezetimibe, and bile acid sequestrants are prevalent, newer treatments like PCSK9 inhibitors and Inclisiran are emerging. Inclisiran, a small interfering RNA therapy, stands out as it requires only two injections per year and achieves a sustained LDL-C reduction of over 50%, with benefits extending to total cholesterol and non-HDL-C. Phase III trials, including the ORION studies, have demonstrated its long-term efficacy and safety, showing consistent LDL-C reductions over several months.

In clinical practice, Inclisiran has proven especially effective in cases of statin intolerance, heterozygous familial hypercholesterolemia, recurrent cardiovascular events, multivessel disease, and young patients with myocardial infarction, achieving LDL-C reductions up to 70-85% within one month of injection and sustained effects for up to eight months. There have been no significant safety concerns reported. The guiding principle in LDL-C management now emphasizes achieving lower levels sooner, maintaining them longer, and recognizing that 'the lower, the better' is integral to improving cardiovascular outcomes.

ROLE OF ARTIFICIAL INTELLIGENCE IN ECHOCARDIOGRAPHY, PRESENT STATUS AND FUTURE PERSPECTIVE.

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Echocardiography is a fundamental diagnostic tool in the field of cardiology. It is essential in most of cardiology patients and has a major role in decision making, classifications and prognosis of patients. Artificial intelligence (AI) as a latest innovation, can help physicians in automation of measurements and interpretation of results. AI builds a computational programe that can perform tasks at par to human interlligence. It mimics human thought process. The use of AI techniques in cardiovascular imaging and decision making will enhance the quality and delivery of care. AI helps in image acquisition, image interpretation, report composition, diagnostic process and prognostication. Artificial intelligence (AI) technologies provide new possibilities for echocardiography to generate accurate, consistent and automated interpretation of echocardiograms, thus potentially reducing the risk of human error. The research suggests it is feasible to apply machine learning models to provide rapid, highly accurate and consistent assessment of echocardiograms, comparable to clinicians. These algorithms are capable of accurately quantifying a wide range of features, such as the severity of valvular heart disease or the ischaemic burden in patients with coronary artery disease. Widespread adoption of obust AI tools in clinical echocardiography practice should follow and have the potential to deliver significant benefits for patient outcome.

MITRAL ANNULAR DISJUNCTION - A NEW DISEASE SPECTRUM - HOW TO MANAGE

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BHU, VARANASI

Introduction

Mitral annular disjunction (MAD) is an abnormal displacement of the mitral valve leaflet onto the left atrial wall and is commonly found in patients with mitral valve prolapse (MVP). The diagnosis is usually made by transthoracic echocardiography (TTE) although findings can be subtle and further cardiac imaging may be necessary. MAD has been associated with a risk of malignant ventricular arrhythmias and sudden cardiac death, therefore recognition of this diagnosis and risk stratification are highly important.

Clinical implication

The clinical outcome of MVP is variable and related to complications such as mitral regurgitation, congestive heart failure, infective endocarditis and stroke. The risk of malignant arrhythmias and SCD is low, occurring in 0.2–0.4% of patients with MVP per year(1), and MVP is not routinely considered as a major cause of SCD.

Diagnosis and multimodality imaging

The diagnosis of MAD is made with cardiac imaging which can be done by transthoracic echocardiography (TTE),transesophageal echocardiography (TEE), cardiac computedtomography (CT) or cardiac magnetic resonance imaging (CMR). It is defined as the absence of myocardium during systole between the MVannulus and the adjacent segment of the left ventricular wall.

Therapies

Patients with significant valvular disease, heart failure or arrhythmias should be treated according to standard AHA/ACC guidelines, however there is little data studying the use of medical therapy such as beta-blockers or

antiarrhythmics in patients with MVP and MAD. Although there are no guidelines regarding catheter ablation in MVP and MAD, this can be performed in patients with symptomatic, drug refractory ventricular arrhythmias.The role of primary prevention ICD in MVP and MAD is unclear. Surgical mitral valve repair or replacement is indicated in patients with MVP and severe MR who are symptomatic or asymptomatic who meetcertain echocardiographic criteria(2).Mitral transcatheter edge-to-edge repair (TEER) with the MitraClip device was approved in the United States in 2013 for the treatment of primary MR.

Conclusions

Mitral valve prolapse and mitral annular disjunction are becoming increasingly recognized as important phenomena that can lead to malignant ventricular arrhythmias and sudden cardiac death.MAD is a common finding in patients with MVP and most of the prior studies have been done in MVP populations. Given that MAD has been recently shown to be arrhythmogenic even in the absence of MVP, therefore recognition of this diagnosis and risk stratification are highly important.

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RESISTANT HYPERTENSION: WHAT IS NEW IN DIAGNOSIS AND MANAGEMENT

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Resistant hypertension (RH) is a complex condition defined by persistently elevated blood pressure despite the optimal use of three or more antihypertensive agents, including a diuretic. Distinguishing true RH from pseudo-resistant hypertension is essential, requiring careful evaluation of factors such as improper blood pressure measurement, medication non-adherence, ruling out white coat hypertension and screening for causes of secondary hypertension (especially primary aldosteronism).

Accurate diagnosis involves standardized blood pressure measurement techniques, including automated office blood pressure (AOBP), home blood pressure monitoring (HBPM), and 24-hour ambulatory blood pressure monitoring (ABPM). Strategies to assess non-adherence and screen for secondary hypertension play a crucial role in refining treatment approaches.

Management includes lifestyle modifications, such as salt restriction and eliminating interfering substances. Pharmacological strategies remain central, with established treatments like spironolactone supported by studies such as PATHWAY-2 and ReHOT. Additionally, newer agents are expanding treatment options. Angiotensin receptor neprilysin inhibitors (ARNIs), aldosterone synthase inhibitors (e.g., Baxdrostat from the BrigHTN trial), and non-steroidal mineralocorticoid receptor antagonists (MRAs) like Ocedurenone (KBP-5074) are showing promise. The ESAX-HTN study has also highlighted the efficacy of Esaxerenone in blood pressure control. Angiotensinogen synthesis inhibitors and the endothelin receptor antagonist Aprocitentan (PRECISION trial) represent further

advancements.

For patients with uncontrolled RH despite pharmacotherapy, device-based treatments like renal denervation (RDN) offer an alternative. Recent developments in catheter systems and procedural refinements have demonstrated improved efficacy in second-generation sham-controlled trials. With these evolving therapies, RH management is advancing toward a more personalized, effective approach, integrating novel pharmacological and interventional strategies to improve blood pressure control and cardiovascular outcomes.

DYSLIPIDAEMIA MANAGEMENT IN PREGNANT PATIENTS: AN UPDATE

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Lipid Management During Pregnancy

- Lipid and lipoprotein levels naturally rise during pregnancy, peaking in the second trimester to support fetal development. Women with pre-existing dyslipidemia experience greater increases, and discontinuing lipid-lowering therapy can elevate cardiovascular risk.

Pre-Conception & Lipid Screening

- Patients with dyslipidemia should consult cardiologists, dietitians, and geneticists pre-conception to assess the risks of stopping treatment. Lipid monitoring during pregnancy is recommended, though optimal timing remains unclear. Routine prenatal lipid testing can aid early diagnosis.

Management of Dyslipidemia in Pregnancy

- Lifestyle Interventions: Dietary changes and regular exercise should be prioritized. A meta-analysis found these interventions reduced gestational weight gain without adverse effects.

Pharmacological Interventions:

- Bile Acid Sequestrants (BAS): The only approved lipid-lowering drugs for pregnancy, BAS lower LDL-C but may impair fat-soluble vitamin absorption.
- Omega-3 Fatty Acids: Safely reduce triglycerides but may increase the risk of large-for-gestational-age babies.
- Statins: Generally contraindicated due to potential fetal harm. However, they may be considered for high-risk patients, such as those with homozygous familial hypercholesterolemia (HoFH). Decisions should involve a multidisciplinary team.

Fibrates & Ezetimibe: Not officially approved, though fibrates may be used in the second trimester if benefits outweigh risks. PCSK9 inhibitors, bempedoic acid, and inclisiran lack safety data.

- Lipoprotein Apheresis: A safe option for severe hypercholesterolemia or hypertriglyceridemia, though its availability is limited.

Monitoring & Postpartum Care

- Regular lipid monitoring, blood pressure, and glucose checks are essential. Postpartum, lipid levels typically normalize, but high-risk women should undergo continued cardiovascular monitoring.

Conclusion

- Given rising maternal age and ASCVD risk, lipid management in pregnancy requires reassessment. While most lipid-lowering drugs are discontinued, statins may be considered in select high-risk cases with close medical supervision.

LONG TERM BETA BLOCKER POST MI - TO INTERRUPT OR TO CONTINUE

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Beta blockers are a crucial part of secondary prevention after acute myocardial infarction to attenuate sympathetic activation, thereby reducing myocardial oxygen consumption, preventing fatal arrhythmias and limiting adverse cardiac remodelling.The clinical benefits of using beta blockers in patients with reduced left ventricular ejection fraction after the development of AMI are well-established. However, evidence of the therapeutic effects of beta blockers in patients with AMI without heart failure or LV systolic dysfunction is relatively sparse. More importantly, most existing evidence for the benefits of beta blockers in patients with AMI was generated when primary percutaneous coronary intervention was not the standard of care for AMI. Given that appropriate revascularisation improves prognosis by increasing myocardial salvage, reducing infarct size and decreasing the risk of arrhythmia, the role of beta blockers in AMI needs to be redefined in contemporary practice. Although the current guidelines recommend the long-term use of beta blockers in all patients with AMI without contraindications to beta blocker therapy, the optimal duration of beta blocker treatment after stabilised AMI without reduced LVEF or HF remains uncertain. Theoretically, discontinuing beta blockers in survivors of AMI without a reduced LVEF or HF may prevent unnecessary overtreatment, save medical costs, limit potential side effects and improve quality of life or adherence to other medications. In this regard, some observational studies have been conducted to evaluate the long-term maintenance effects of beta blockers beyond 1 year after AMI, but the non-randomised nature of such studies limits their conclusions due to the presence of selection bias.This talk will throw some light on the same.