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Advances in Obesity and Lipid Management in CVD

(Cutting Edge Strategies by Multispeciality Lens)

A Textbook of Cardiology

Editors- in-Chief

HK Chopra (India) • Navin C Nanda (USA) • Jagat Narula (USA)
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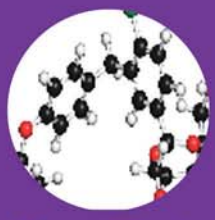
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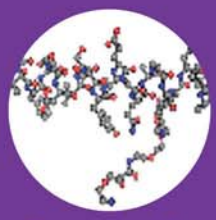
Dapagliflozin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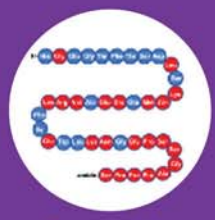
Tirzepatide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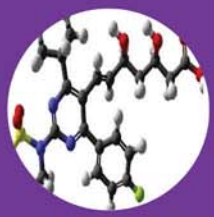
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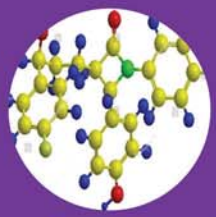

Ezetimibe

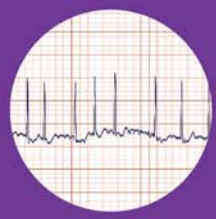

Bempedoic acid


PCSK9i

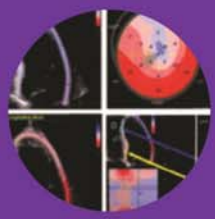

Inclisiran

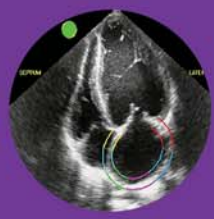




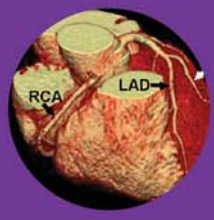




















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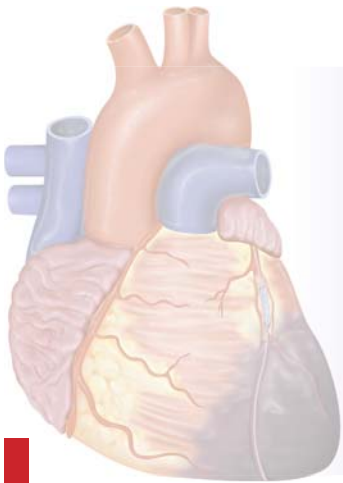

Advances in Obesity and Lipid Management in Cardiovascular Diseases: Cardiac Anesthetist Perspective

Poonam Malhotra Kapoor

JAYPEE BROTHERS

INTRODUCTION

Obesity and dyslipidemia are tightly coupled drivers of atherosclerotic cardiovascular disease (ASCVD) and of perioperative risk in cardiac surgery. Over the last 5 years, the field has shifted from largely lifestyle-and-statin centric care to a multimodal pharmacologic and procedural armamentarium: High-potency statins remain foundational, but novel antiobesity agents [glucagon-like peptide-1 (GLP-1) receptor agonists and dual incretin agonists], second-line LDL-lowering therapies (PCSK9 monoclonal antibodies, siRNA agents such as inclisiran), and nonstatin small molecules (bempedoic acid)—plus agents targeted at triglyceride-related risk (icosapent ethyl)—have changed how we think about long-term risk reduction and short-term perioperative physiology. These advances have implications for preoperative optimization, intraoperative management, and early ICU care.[1]

PATHOPHYSIOLOGIC RATIONALE BACKGROUND THAT IS IMPORTANT

Adiposity as a multimodal stressor: Adipose tissue in obesity is not inert: visceral adiposity produces systemic inflammation, hypercoagulability, and adverse myocardial remodeling; it also predisposes to difficult airway, reduced functional residual capacity, obstructive sleep apnea (OSA), and altered pharmacokinetics of anesthetic agents. These mechanisms explain the increased incidence of postoperative atrial fibrillation, low cardiac output, and pulmonary complications in obese cardiac surgery patients.[2]

Targeting Obesity in Cardiovascular Disease Management: Cardiac Adipose Tissue Is a Real Biomarker!

There are different types of adipose tissue, including cardiac adipose tissue, which comprises epicardial and pericardial (or paracardiac) fractions. Epicardial adipose tissue (EAT) is unique due to its proximity to the heart and a distinct gene expression profile compared to other adipose depots, such as visceral and subcutaneous fat. EAT plays a crucial role in the development and progression of cardiovascular diseases (CVDs) with high morbidity and mortality, acting both as a metabolic and inflammatory mediator.

Cardiac adipose tissue, particularly EAT, is a key player in cardiometabolic disease. Understanding its pathophysiological role and incorporating imaging tools to evaluate EAT may enhance cardiovascular risk stratification and disease management.[3]

In a position statement by five medical societies, management of obesity and prevention of CVD and obesity-associated complications following cardiovascular risk assessment using the Predicting Risk of CVD Events risk score, individuals with obesity or overweight will be stratified according to their 10-year risk of developing atherosclerotic disease (low, moderate, or high) and heart failure (high-risk). Antiobesity treatment will then be guided by the best evidence-based recommendations designed to address excess adiposity and reduce associated complications, as shown in **Table 1**.

TABLE 1: Cardiovascular risk assessment in individuals with overweight or obesity.

High—20% at 10 years	• Established coronary artery disease, myocardial infarction (MI), ischemic stroke or transient ischemic attack (TIA), peripheral artery disease (PAD), arterial revascularization in any territory • Primary prevention with total CV risk by PREVENT score—20% at 10 years • Type 2 diabetes duration >10 years. • Chronic kidney disease stage 3b/4/5 • Coronary artery calcium (CAC) score >100 Agatston units (AU). (without diabetes) or >10 AU (with diabetes), Lp(a) >50 mg/dL or >125. nmol/L, familial hypercholesterolemia (FH), or LDL-C >190 mg/dL
High HF risk—20% at 10 years	• BMI >40 kg/m^2, even if asymptomatic • Individuals with obesity, plus diabetes and hypertension • Severe obstructive sleep apnea (OSA) • Atrial fibrillation • Chronic kidney disease stage 3b/4/5 • Elevated NT-proBNP or BNP • PREVENT HF risk score—20% at 10 years • Established ASCVD • Symptoms suggestive of HF

RESIDUAL LIPID RISK AND PLAQUES VULNERABLE TO PERIOPERATIVE STRESS

Low-density lipoprotein cholesterol (LDL-C) lowering reduces plaque progression and stabilizes lesions; inflammation and triglyceride-rich lipoproteins remain residual risk domains even when LDL is controlled. New agents lower these risks by complementary mechanisms (PCSK9 pathway, ACL-inhibition, and RNA interference) and—in trials—reduce events beyond statin monotherapy. Understanding the mechanism helps us anticipate effects (e.g., on inflammation, wound healing, and rare hepatic effects) that may be clinically relevant preoperatively.[4]

EPIDEMIOLOGY OF OBESITY IN HEART FAILURE

Avoidance of excess adiposity throughout the lifespan is key to the prevention of incident Heart Failure, particularly HFpEF. The "Heart failure obesity survival paradox" in part represents the negative implications of unintentional weight loss. Obesity treatments offer opportunities for improved heart failure symptom burden, functional capacity, quality of life, and fewer hospitalizations.[5]

MAJOR THERAPEUTIC ADVANCES–WHAT CHANGED AND WHY IT MATTERS?

Antiobesity Pharmacotherapy with Cardiovascular Benefit

Semaglutide (Weekly 2.4 mg)

The SELECT cardiovascular outcomes trial showed a ~20% relative reduction in major adverse cardiovascular events (MACE) in overweight/obese patients with established CVD (without diabetes), together with sustained weight loss and renal benefits. For the anesthetist, this class reduces body weight, improves hemodynamics and metabolic profile long-term, but has perioperative considerations.[1]

Tirzepatide (Dual Glucagon-like Peptide/Glucagon-like Peptide-1 Agonist)

Tirzepatide produces larger weight loss than first-generation GLP-1 RAs in obesity trials (SURMOUNT series) and favorable cardiometabolic improvements; cardiovascular outcome evidence is emerging (meta-analyses and real-world studies suggest safety and possible benefit), but large CVOTs are ongoing. From an anesthetic viewpoint, tirzepatide's potent weight loss and glycemic effects may change drug dosing, fluid distribution, and stress responses.[6]

Why are these clinically relevant? These drugs are shifting preoperative profiles (fewer BMI-related complications long term) but introduce short-term issues: Delayed gastric emptying, nausea, increased gallstone risk, and the need to manage hypoglycemia risk and changes in drug absorption and volume of distribution.

NEW AND REPURPOSED LIPID-LOWERING THERAPIES

Bempedoic Acid (Adenosine Triphosphate Citrate Lyase Inhibitor)

The CLEAR Outcomes trial in statin-intolerant patients demonstrated a statistically significant reduction in MACE versus placebo, with LDL lowering and favorable safety signals in the trial population. This provides an oral non-statin option for patients' intolerance of statins.[7]

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Proprotein Convertase Subtilisin/Kexin Type 9 Inhibitors (Monoclonal Antibodies) and Ribonucleic Acid Therapeutics

Monoclonal PCSK9 inhibitors (evolocumab and alirocumab) remain in potent LDL-lowerers with proven cardiovascular event reduction in high-risk populations. Inclisiran (siRNA targeting PCSK9) offers twice-yearly dosing with meaningful LDL-C reductions; large outcomes trials (ORION/ORION-4/HPS-4) and recent data on broader populations have expanded clinical interest, though outcome-level evidence is still accruing.[8]

Icosapent Ethyl (High-dose Eicosapentaenoic Acid, Reduction of Cardiovascular Events with Icosapent Ethyl–Intervention Trial)

Reduces ischemic events in selected patients with elevated triglycerides despite statin therapy and remains a consideration for residual triglyceride-driven risk.[9]

Why is clinically relevant? For the cardiac anesthetist, these agents alter the patient's baseline risk and inflammatory milieu; they can influence perioperative LDL/inflammatory markers and, rarely, have hepatic or hematologic side effects to consider in perioperative laboratories/monitoring. Evidence for perioperative continuation is limited for the newer agents, but continuation of statins perioperatively is supported by guidelines and associated with improved short-term outcomes.

INTENSIVE CARE UNIT CONSIDERATIONS IN OBESITY AND DYSLIPIDEMIA

Metabolic Recalibration

If a patient is on GLP-1 RA or tirzepatide, document start date, dose, GI tolerance, and glycemic trends. For patients actively losing weight on these agents, reassess airway and pulmonary function (OSA screening), nutritional status, and liver function. GLP-1 agents commonly cause nausea and delay gastric emptying—treat as at-risk for aspirations.[1]

Lipid Therapy

Continue chronic statin therapy through the perioperative period (guideline supported); initiation of statin therapy at least 1–2 weeks of preoperative is reasonable for high-risk vascular patients. For PCSK9 or inclisiran recipients, evidence to guide perioperative continuation is limited—decisions should be multidisciplinary (cardiology, surgery, and anesthesia) and individualized; there is no class-wide recommendation to stop these agents before cardiac surgery at present.[10]

Nutritional and Adiposity Indices

Consider combining adiposity-nutritional indices (CANI) as predictors of MACCE after CABG (evidence from cardiac critical care literature); use such indices to inform risk discussion and ICU planning.[11]

Glycemia and Nutrition

Glucagon-like peptide-1-class drugs reduce gastric emptying and may increase nausea postoperatively—anticipate altered enteral feeding tolerance and avoid sudden discontinuation that could destabilize glycemic control. If the patient is nil per oral and requires intensive insulin therapy, follow institutional insulin protocols; coordinate with endocrinology for bridging.

Infectious Risk and Wound Healing

Weight loss and altered nutritional state (rapid or ongoing) can impact wound healing. Monitor for bile-related complications (increased gallstone risk with rapid weight loss) and pancreatitis if clinically suspected in patients on GLP-1 or dual agonists.

Continuation of Secondary Prevention

Restart statins early postoperatively unless contraindicated; where PCSK9 monoclonal therapy or inclisiran has been used, coordinate timing of dosing with the cardiology team—no strong evidence yet to mandate routine perioperative withholding.

Airway and Ventilation

Expect decreased FRC, rapid desaturation and increased risk of difficult mask ventilation/intubation in higher BMI; have an OSA plan, use recruitment strategies, and protective ventilation (lower tidal volume, higher PEEP titrated to physiology).[2]

Hemodynamic Effects

Rapid weight loss or metabolic changes from antiobesity drugs may lead to altered vasomotor tone and myocardial energetics—be vigilant for exaggerated BP and HR responses during induction and rewarming. Adjust

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dosages carefully and monitor serum electrolytes and glucose intraoperatively.

Coagulation and Inflammation

Aggressive LDL-lowering and agents that modulate inflammation may blunt classic inflammatory surges, but data on immediate pro/anticoagulant effects peri-CABG are limited. Use standard point-of-care coagulation monitoring (ROTEM/TEG) as always.

DIAGNOSIS OF OBESITY

Although BMI is an inexpensive, easily acquired, and readily reproducible metric that is strongly embedded in research and clinical practice, significant limitations exist in the detection of excess adiposity, the location of adiposity, and applicability to diverse populations. To determine a diagnosis of clinical obesity in individuals with BMI <35 kg/m^2, direct assessment of excess adiposity with an anthropometric criterion [e.g., waist circumference (the most readily obtainable), waist-to-hip ratio, or waist-to-height ratio] or body composition assessment (e.g., dual X-ray absorptiometry, when available) may be used. Clinicians should be aware that obesity remains a stigmatized condition and that some individuals may experience discomfort with being weighed, abdominally measured, or talking about weight during a medical visit.

CHALLENGES OF THE DIAGNOSIS OF HEART FAILURE IN INDIVIDUALS WITH OBESITY

Because individuals with obesity have lower NP concentrations, lower thresholds are used in those who have obesity and exertional dyspnea to avoid underdiagnosis of HF in this population, although specific thresholds are not currently established. For individuals with HF and obesity, monitoring for T2DM, hypertension, atrial fibrillation, sleep disordered breathing, and objective evidence of exercise intolerance can identify the need for targeted interventions.

MANAGEMENT OF OBESITY IN HEART FAILURE

Behavioral changes aimed at intentional weight loss are appropriate to attempt for individuals with obesity because even modest changes in body weight can result in improvements in risk of cardiovascular events, although weight loss is often unsustainable. Exercise can improve functional status in individuals with HFpEF.

ROLE OF ANTIOBESITY MEDICATIONS IN HEART FAILURE WITH PRESERVED EJECTION FRACTION WITH OBESITY

The STEP-HFpEF program and SUMMIT trial show that, in people with BMI $30 kg/m^2 and HF with EF $45% (semaglutide) and EF $50% (tirzepatide), weight loss is associated with improvements in symptoms and functional capacity. Insufficient evidence exists to date to confidently conclude that semaglutide and tirzepatide reduce HF events in individuals with HFpEF and obesity (with stronger evidence for tirzepatide), although exploratory analysis indicates favorable changes in biomarkers and imaging parameters, suggesting potential distinct mechanistic advantages outside of weight loss.

During early-phase gradual dose escalation of semaglutide or tirzepatide, which occurs every 4 weeks, monitor kidney function and electrolytes with adjustment of diuretics, antihypertensive agents, and antihyperglycemic agents as indicated, particularly if gastrointestinal adverse effects are prominent.

INVASIVE INTERVENTIONS

For individuals with HF and obesity, MBS appears effective for intentional weight loss and potentially to reduce the risk of HF events, including hospitalization for HF and death, although these possibilities are based only on data from observational studies. Individuals with HF who are undergoing MBS have an increased risk of postoperative cardiovascular morbidity and death, suggesting the need for preoperative optimization and perioperative care by clinicians with expertise in HF management.

EMERGING QUESTIONS AND AREAS FOR FUTURE STUDY (BRIEF)

- *Class effect vs agent specificity for CV protection:* SELECT (semaglutide) and SURMOUNT/SURPASS (tirzepatide) signal important cardio metabolic benefits, but head-to-head and mechanistic work to separate weight-mediated from pleiotropic effects is ongoing.[13]
- *Perioperative trials:* Trials that test perioperative continuation vs temporary suspension of novel lipid-lowering agents and GLP-1/dual agonists are sparse; cardiac surgery is an ideal setting to study whether rapid LDL/inflammatory modulation improves myocardial protection and short-term outcomes.
- *Implementation and equity:* Access, cost, and real-world adherence to twice-yearly inclisiran or expensive

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GLP-1 therapy will shape outcomes; real-world comparative effectiveness studies are emerging.

CONCLUSION

Obesity confers increased risks of HF, coronary artery disease, and stroke[12] and weight loss can reduce CVD risk. Given emerging evidence of the benefits of semaglutide and tirzepatide in individuals with HFpEF and obesity in concert with healthy behavioral interventions, clinicians should be aware of optimal diagnosis, risk assessment, and management of obesity in individuals with HF. An essential future priority will be providing equitable access through policies and systems to ensure that individuals with adverse SDOH have access to evidence-based AOM, particularly so that we can avoid the unintended consequence of further exacerbating health inequities. With accurate evaluation of obesity as well as administration and monitoring of safe and effective interventions, clinicians may improve quality of life and functional capacity and potentially reduce HF events in individuals living with HF and obesity.

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It is the first publication of its kind in the form of a 14th focused textbook of cardiology from India to the world. More than 300 contributors from India and abroad have contributed their prestigious chapters in this book, focusing on every aspect of obesity and lipid management including advances in management of obesity-inflicted morbidity and mortality such as cardiovascular disease (CVD), chronic kidney disease (CKD), diabetes mellitus (DM), and heart failure (HF), and cardiometabolic modulation by GLP-1 receptor agonists (GLP-1RAs) such as Semaglutide and Tirzepatide and lipid management with statins, ezetimibe, bempedoic acid, PCSK9i, inclisiran apheresis, and gene therapy. The efficacy of drug therapy by biomarkers, echocardiography, and multimodality imaging is very well elucidated and has tremendous innovative and research potential of understanding advances in obesity and lipid management for the undergraduates, postgraduates, practicing cardiologists, endocrinologists, nephrologists, neurologists, critical care specialists, pulmonologists, pediatricians, pediatric surgeons, physicians, cardiovascular surgeons, imaging experts, nutritionists, etc. The book includes 23 sections and 172 chapters. The book is being dedicated to India and the world.



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