LUCKNOW CARDICON

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Atal Bihari Vajpayee Scientific Convention Center, Lucknow

NEWSLETTER

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FEB 2026

From the Desk of the Editor

Dr. Roopali Khanna

Dear Readers,

We are pleased to launch the new issue of the Newsletter in February, 2026. This issue provides an overview of recent topics in the field of cardiology.

This issue features a diverse range of topics in the field of Hypertension, Coronary Artery Disease, Dyslipidemia, and Heart Failure, including arrhythmias, advanced structural interventions, and challenges in coronary and pediatric interventions. Our contributors have worked tirelessly to bring you relevant and resonant content.

Thank you for your continued support. I hope this issue provides you with knowledge, inspiration, and perhaps even a new perspective.

We also anticipate an excellent meeting organized by Dr Akshyaya Pradhan, CARDICON 2026, 13th & 14th February, 2026, Lucknow

Editor
Dr. Roopali Khanna
Lucknow

FEB 2026

Overview of the Scientific Program

Dear Colleagues,

It gives us immense pleasure and pride to invite you to CARDICON 2026, the 31st Annual Conference of the U.P. Chapter of the Cardiological Society of India (UP-CSI), scheduled to be held on 13th & 14th February 2026 at the Atal Bihari Vajpayee Scientific Convention Centre, KGMU, Lucknow. This flagship academic event continues to be one of the most important cardiology meetings of the state, bringing together the finest minds in cardiovascular medicine.

The scientific program has been meticulously designed to address the evolving landscape of contemporary cardiology practice. Eminent regional and national faculty will deliberate on key clinical and interventional domains, ensuring a balanced blend of evidence-based medicine, real-world experience, and future-ready innovations.

The program comprehensively covers coronary artery disease, heart failure, hypertension, lipidology, arrhythmias and ECG, valvular heart disease, congenital heart disease, cardiometabolic disorders, cardiovascular pharmacology, women and heart disease, and the growing role of artificial intelligence in cardiology. Special emphasis has been laid on practical decision-making, challenging case discussions, and guideline-directed yet individualized patient care.

To further enrich the academic experience, the agenda includes ECG quizzes for residents and physicians, DM Cardiology quizzes for trainees, and dedicated educational sessions for CV technicians and paramedical staff, making CARDICON 2026 inclusive, interactive, and engaging for all sections of the cardiology fraternity.

The entire Scientific Committee and Organizing Committee sincerely hope that this academic feast will be clinically meaningful, academically stimulating, and inspiring, fostering new ideas, collaborations, and research initiatives.

We extend a warm and heartfelt welcome to you all and look forward to your enthusiastic participation in CARDICON 2026.

Dr. Umeshwar Pandey
President Elect, UP-CSI

ETHICS IN CARDIOLOGY PRACTICE :

FEB 2026

Ajay Kr Pandey
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Ethics in cardiology practice is an essential aspect of delivering high-quality patient care while navigating the complex landscape of medical innovations, patient rights, and professional responsibilities. Ethics is an inherent& inseperable part of clinical medicine. Physicians have an ethical obligation a) to take care of patient's benefit b)to respect values & preferences of patient c)to minimize or avoid harm(1) .

Medical practice & scientific research needs application of medical ethics. As in all areas of medicine, cardiology is driven by the "four fundamental moral principles" formulated by Tom Beauchamp & James Childress (2)

The four principles are :

1. Respect for Autonomy -the patient has the right to refuse or choose their treatment.
2. Beneficence - to act in best interest of patient .
3. Non-maleficence – to not be the cause of harm, Also, 'Utility" -to promote more good than harm.
4. Justice- concerns the distribution of scarce health resources & the decision of who gets what treatment.

Autonomy

The principal of Autonomy, observes the right of an individual to self determination (3) . this refers to the patient's right to make informed decisions about their healthcare. In cardiology, this principle is paramount due to the often complex diagnostic and therapeutic options available. Patients must be adequately informed about their condition, the available treatment modalities, their risks and benefits, and potential outcomes. Shared decision-making is encouraged, where patients engage in discussions with their healthcare providers to make choices that align with their values and preferences.

However, respecting autonomy can sometimes lead to ethical dilemmas, especially when patients refuse life-saving interventions or adhere to non-evidence-based alternatives. In these cases, cardiologists must balance respect for patient choices with the obligation to provide the best possible care. It becomes essential to foster a trusting relationship, ensuring patients feel understood and respected, even when their decisions diverge from medical recommendations.

Beneficence and Non-Maleficence

Beneficence involves acting in the best interests of the patient,(4) while non-maleficence focuses on the obligation to avoid harm.(5) In cardiology, this translates to delivering treatments that offer genuine benefits while minimizing potential risks. The rapid advancement of cardiac interventions and technologies, such as catheter-based procedures, implantable devices, and pharmacologic therapies, poses challenges in ensuring that these techniques are appropriate for each patient.

Cardiologists must stay informed about the latest research and clinical guidelines to offer evidence-based recommendations. Moreover, they should consider individual patient factors—such as age, comorbidities, and personal preferences—when assessing the appropriateness of a treatment plan.

Another vital consideration is the potential for over-treatment in cardiology, especially when aggressive interventions may not improve quality of life. The ethical challenge lies in determining when to recommend invasive procedures versus conservative management, always prioritizing the overall well-being of the patient.

Justice

Justice in healthcare relates to the fair distribution of resources and the equitable treatment of patients. In cardiology, disparities in access to care can be significant, influenced by socioeconomic factors, geographic location, and racial or ethnic backgrounds. Cardiologists must advocate for policies that promote health equity, ensuring that all patients have access to necessary cardiac care, regardless of their circumstances.

Moreover, cardiology practice should consider the allocation of resources in terms of cost-effectiveness and the sustainability of healthcare systems. In an era of rising healthcare costs, ethical practice requires balancing individual patient needs with broader public health considerations.

Patient Enrollment in Clinical Trials :

An emerging ethical consideration in cardiology is the recruitment and enrollment of patients in clinical trials. Ensuring that patients understand the purpose, risks, and potential benefits of participating in research is critical. Transparency in the informed consent process is vital, and efforts should be made to include diverse populations in clinical studies to enhance the applicability of the findings.

Additionally, the ethical principle of justice requires that vulnerable populations are not exploited for research purposes but rather are afforded the same opportunities for benefiting from advancements in cardiac care.

Ethics Committees :

Often, simple communication is not enough to resolve conflicts, and a hospital ethics committee must convene to decide on complex matters. There presence is mandatory for smooth functioning of clinical research & to provide balance.

Conclusion :

In conclusion, ethics in cardiology practice encompasses a multifaceted approach to patient care, emphasizing the principles of autonomy, beneficence, non-maleficence, and justice. As cardiology continues to evolve with new technologies and therapies, practitioners must critically reflect on their ethical obligations to their patients, the healthcare system, and society as a whole. By fostering transparent communication, embracing shared decision-making, and advocating for equitable access to care, cardiologists can ensure that they practice ethically and effectively in the complex landscape of cardiovascular medicine.

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Lp(a) – Small Molecule, Big Trouble

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Lipoprotein(a), abbreviated Lp(a), is increasingly recognised as one of the most important, genetically determined, and underappreciated contributors to atherosclerotic cardiovascular disease (ASCVD). Although it appears "small" in routine clinical discussions and is often absent from standard lipid panels, its clinical impact is undeniably large—hence the phrase: **"Lp(a) – small molecule, big trouble."**

What is Lp(a)?
Lp(a) is a unique lipoprotein particle synthesised in the **liver,** structurally composed of an **LDL-like particle containing ApoB100,** covalently linked via a disulfide bond to a distinctive protein called **apolipoprotein(a) [apo(a)],** which resembles plasminogen. This hybrid design poses a dual threat: it behaves like LDL by depositing cholesterol in the vessel wall, and simultaneously promotes thrombosis through its apo(a) component.

Why is Lp(a) dangerous?
Lp(a) is not just another "cholesterol parameter." It is a powerful **independent risk factor** for **coronary artery disease (CAD), stroke,** and **peripheral arterial disease (PAD).** Elevated levels are found in approximately **20–25% of the global population,** making it a highly prevalent cardiovascular risk amplifier.

Unlike standard LDL particles, **each Lp(a) particle is estimated to be about 6 times more atherogenic than each LDL particle,** making even moderately elevated levels clinically meaningful.

The genetics trap: lifestyle won't save you
Perhaps the most frustrating aspect of Lp(a) is that its levels are **primarily genetically determined,** inherited in a **Mendelian fashion,** and show **minimal influence from lifestyle, diet, or environmental factors.** This explains why many young patients with premature myocardial infarction may present with "acceptable" LDL-C values but still experience aggressive atherosclerosis—Lp(a) often hides in plain sight.

Additionally, there are **ethnic differences,** with higher Lp(a) concentrations frequently observed in populations of **African descent.**

Mechanisms: atherosclerosis + thrombosis in one package
Lp(a) creates vascular damage through multiple interlinked pathways:

1. **Atherogenic burden (LDL component):** Like LDL, it enters the arterial wall, where it can undergo oxidation and contribute to **foam cell formation** and plaque development.
2. **Pro-inflammatory activity:** Lp(a) carries **oxidised phospholipids,** triggering endothelial dysfunction and chronic vascular inflammation.
3. **Pro-thrombotic potential (apo(a) component):** Apo(a) resembles plasminogen and promotes **atherothrombosis,** impairing normal fibrinolysis and tipping the balance toward clot formation.

Thus, Lp(a) not only accelerates plaque formation but also increases the likelihood of plaque disruption and thrombosis—making it a potent driver of acute coronary events.

Risk underestimation: the silent amplifier
High Lp(a) can massively underestimate cardiovascular risk if not measured. For example, a markedly elevated Lp(a) (around 150 mg/dL) can lead to an approximately 2.7-fold increase in ASCVD risk, and may shift lifetime risk dramatically upward. This is why many "unexpected" events happen despite "controlled" LDL.

Testing: from selective screening to universal once-in-a-lifetime
Recommendations around Lp(a) testing have evolved significantly. Earlier guidance focused on testing only in high-risk groups, such as:

- family history of premature ASCVD
- familial hypercholesterolemia
- premature personal ASCVD
- aortic stenosis

However, modern guidelines increasingly support a **once-in-a-lifetime Lp(a) measurement in all adults,** as it is simple, prevents delayed recognition, and enables earlier preventive strategies.

Treatment: current gaps, future hope
The major clinical challenge is that **traditional lipid-lowering therapies do not effectively target Lp(a).** Statins may modestly increase Lp(a) levels and do not meaningfully reduce them. Niacin lowers Lp(a) by about 20% but lacks outcome benefit and has tolerability issues. Ezetimibe and bempedoic acid provide minimal to no clinically relevant Lp(a) lowering.

Currently, the most consistent available options are:

- **PCSK9** inhibitors, lowering Lp(a) roughly **20–30%**
- **Lipoprotein apheresis,** reserved for select indications

The real excitement lies in emerging therapies targeting **LPA gene expression,** including antisense oligonucleotides and siRNA therapies such as **pelacarsen, olpasiran, zerlasiran, and lepodisiran,** showing profound reductions in Lp(a). There is also promise with **muvalaplin,** an oral small-molecule inhibitor of Lp(a) formation.

Conclusion
Lp(a) represents a classic modern cardiology paradox: a highly prevalent and powerful risk factor that remains underdiagnosed, under-measured, and undertreated. It is genetically encoded, biologically aggressive, and clinically deceptive—truly a **small molecule with big trouble.** The most practical step today is simple: **measure Lp(a), reclassify risk appropriately, and intensify management of modifiable risk factors** while preparing for the coming era of targeted Lp(a)-lowering therapies.

The Paradigm Shift in Post-PCI Antithrombotic Management: A Comprehensive Analysis of Dual Antiplatelet therapy abbreviation and the Emergence of P2Y12 inhibitor Monotherapy

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The Legacy of the 12-Month Standard

The history of interventional cardiology is, in many ways, a history of battling thrombosis. In the early era of balloon angioplasty and bare-metal stents (BMS), the primary adversary was abrupt vessel closure and acute stent thrombosis. The introduction of aspirin combined with ticlopidine, and later clopidogrel, revolutionized the field, significantly reducing these catastrophic events. The subsequent advent of first-generation drug-eluting stents (DES), while solving the problem of restenosis, introduced a new fear: late and very late stent thrombosis. This concern cemented the "12-month DAPT" duration into clinical practice, a standard largely implied from the design of seminal trials like **CURE** (Clopidogrel in Unstable Angina to Prevent Recurrent Events) rather than one derived from direct duration-testing studies.

For years, the cardiovascular community operated under the premise that "more is better" regarding platelet inhibition. Guidelines mandated 12 months of DAPT for ACS patients, driven by the desire to mitigate ischemic recurrences. However, as stent technologies evolved from thick-strut stainless steel scaffolds to ultrathin cobalt-chromium platforms with biocompatible or biodegradable polymers, the thrombotic risk profile of the treated vessel began to recede. Concurrently, the introduction of potent P2Y12 inhibitors—prasugrel (TRITON-TIMI 38) and ticagrelor (PLATO)—further suppressed ischemic events but inescapably amplified bleeding risks.

The Decline of Aspirin Monotherapy

A crucial insight from this hierarchy is the downgrading of aspirin monotherapy. The consensus notes that aspirin monotherapy portends a "greater risk of ischemic events than long-term DAPT" while offering "hardly any advantage in terms of bleeding endpoints" when compared to P2Y12 inhibitor monotherapy. This challenges the decades-old practice of lifelong aspirin, suggesting that for many patients, the P2Y12 inhibitor should be the single agent of choice for long-term maintenance.

The "Aspirin-Free" Future: OPTICA and Beyond

If withdrawing aspirin at 1 month is beneficial, could withdrawing it immediately be better? The logical endpoint of this research trajectory is the total elimination of aspirin from the post-PCI regimen.

The OPTICA Study (2023)

The OPTICA (Optical Coherence Tomography-Guided PCI with Single-Antiplatelet Therapy) study was a proof-of-concept pilot trial designed to test this boundary.

- **Protocol:** Patients with non-ST-elevation ACS (NSTE-ACS) underwent OCT-guided PCI. They received a loading dose of a P2Y12 inhibitor (ticagrelor or prasugrel) but no aspirin loading or maintenance dose.
- **Results:** The study demonstrated feasibility with no catastrophic acute thrombotic events in the small cohort (n=75).
- Significance: While too small to change guidelines, OPTICA proves that in a highly controlled environment with perfect stent optimization (via OCT), the mechanical and pharmacological solution (stent + potent P2Y12) suffices without aspirin.

Clinical Guidelines and Practice Implications

The weight of evidence from ULTIMATE-DAPT, T-PASS, and TICO is reshaping international guidelines, moving the field from a rigid, time-based mandate to a personalized, risk-based approach.

ESC Guidelines (2023 Update)

The **2023 ESC Guidelines for the Management of Acute Coronary Syndromes** reflect this evolution:

- **Default:** 12 months DAPT remains the default recommendation (Class I) for the general population to ensure broad applicability.
- **Abbreviation (Class IIa):** In patients who are event-free after 3–6 months and not at high ischemic risk, DAPT should be discontinued in favor of monotherapy.
- **De-escalation (Class IIb):** In HBR patients, switching to P2Y12 monotherapy (specifically ticagrelor or prasugrel) after just 1 month is now a recognized and endorsed strategy.

ACC/AHA Guidelines (2025)

The American guidelines have similarly adopted a Class IIa recommendation for abbreviated DAPT (1–3 months) followed by P2Y12 monotherapy. The emerging 2025 updates are expected to strengthen this recommendation in light of ULTIMATE-DAPT, potentially elevating the 1-month switch to a Class I recommendation for specific HBR subgroups.

Practical Implementation

For the practicing cardiologist, the workflow is shifting:

1. **Assess Bleeding Risk:** Use ARC-HBR or PRECISE-DAPT scores at baseline.
2. **Select Stent & Technique:** Utilize DES and, whenever possible, intravascular imaging (IVUS/OCT) to ensure mechanical perfection.
3. **The "One-Month Challenge":** Treat with DAPT (Aspirin + Ticagrelor) for 1 month.
4. **Re-evaluate:** If the patient has had no ischemic events, no stent thrombosis, and adhered to therapy, **drop the aspirin.** Continue Ticagrelor 90mg BID.

5. **Long-term:** At 12 months, re-assess. Evidence suggests continuing P2Y12 monotherapy might be superior to switching back to aspirin, though cost issues (generic clopidogrel vs. branded/expensive ticagrelor) often dictate a switch to clopidogrel or aspirin monotherapy in chronic phases.

Limitations and Unmet Needs

Despite the enthusiasm, significant gaps remain that prevent the universal adoption of the "1-month" strategy.

Complexity Bias

A recurring criticism of ULTIMATE-DAPT and T-PASS is the relatively low complexity of the enrolled patients.

- **The Gap:** In ULTIMATE-DAPT, ~70% of patients had single-vessel disease. Patients with left main disease, heavily calcified bifurcations requiring rotablation, or chronic total occlusions (CTOs) were largely underrepresented.
- **The Risk:** In these "complex PCI" scenarios, the thrombotic risk is driven by mechanical factors (multiple stents, overlap zones, slow flow) that might require the dual-pathway inhibition of DAPT for a longer duration (e.g., 3–6 months). Applying a 1-month monotherapy strategy to a patient with a crush-stented left main bifurcation remains an extrapolation of data.

The "East Asian Paradox" and Generalizability

Many of the pivotal monotherapy trials (T-PASS, TICO, STOP-DAPT series, ULTIMATE-DAPT) were conducted in East Asian populations (South Korea, Japan, China).

- **The Physiology:** East Asian patients have a well-documented "East Asian Paradox"—they have a lower rate of ischemic events but a higher propensity for bleeding compared to Western populations, despite lower responsiveness to clopidogrel.
- **Implication:** The massive bleeding benefit seen in these trials might be attenuated in Western populations (who are more pro-thrombotic and have higher BMI), although the **TWILIGHT** trial (which included Western sites) supports the general safety of the strategy.

Imaging Penetrance

The reliance on IVUS/OCT in these trials is a double-edged sword. It ensures internal validity (the stents were open) but limits external validity. In centers where angiography alone is used, the rate of suboptimal stent expansion is higher. Withdrawing aspirin at 1 month in a sub-optimally deployed stent could be catastrophic. Thus, the "1-month DAPT" strategy should arguably be linked to a "mandatory imaging" mandate.

Conclusion

The publication of **ULTIMATE-DAPT** and **T-PASS** marks a definitive turning point in interventional cardiology. The era of the "12-month mandatory DAPT" for all ACS patients is ending, replaced by a nuanced, physiology-based approach that prioritizes the reduction of bleeding.

The evidence is now overwhelming that in patients with acute coronary syndromes, **ticagrelor monotherapy** initiated after a stabilization period of 1 month (or even less) provides a superior net clinical benefit compared to conventional DAPT. This strategy reduces major bleeding by approximately 50% without compromising anti-ischemic efficacy. This success is likely driven by the unique pharmacological profile of ticagrelor, which combines potent, reversible P2Y12 inhibition with pleiotropic, adenosine-mediated cardio protection—a "safety net" that clopidogrel and prasugrel do not reliably provide in the monotherapy setting.

While aspirin remains a crucial tool for the acute phase of plaque rupture and procedural management, its role in the maintenance phase is rapidly diminishing. As clinical guidelines evolve to reflect this hierarchy, the future of post-PCI care appears to be increasingly "aspirin-sparing," offering patients a path to protection that no longer demands the price of hemorrhage.

While aspirin remains a crucial tool for the acute phase of plaque rupture and procedural management, its role in the maintenance phase is rapidly diminishing. As clinical guidelines evolve to reflect this hierarchy, the future of post-PCI care appears to be increasingly "aspirin-sparing," offering patients a path to protection that no longer demands the price of hemorrhage.

Suggested Reading

- Khare H, et al. "Insights on DAPT Abbreviation and De-escalation from ULTIMATE-DAPT and Related Trials: Are We Heading Toward an Aspirin-Free Strategy?" Am J ````` Cardiovasc Drugs, 2025.
- ULTIMATE-DAPT Trial results, statistics, and editorial critiques (Lancet 2024).
- T-PASS Trial results and design (Circulation 2024).
- European Society of Cardiology (ESC) and ACC/AHA Guidelines (2023, 2025).
- EAPCI/ACVC/EAPC Joint Clinical Consensus Statement (2022) on antiplatelet hierarchy.
- Pleiotropic effects of ticagrelor (Adenosine/ENT1 mechanisms).
- OPTICA Study (EuroIntervention 2023).
- STOP-DAPT-3 and STOP-DAPT-2-ACS trial details.

Summary of Trials Investigating Abbreviated DAPT after PCI

Trial	Cohort	Intervention	Comparator Group	Primary Endpoint	Results	Remarks
STOPDAPT-2 (2019)	1525 PCI pts with DES; stable CAD & ACS; low bleeding risk	1-month DAPT → clopidogrel monotherapy	12-month DAPT	Death, MI, stent thrombosis, stroke, TIMI bleeding	1-month DAPT superior (less bleeding)	Lower primary endpoint with short DAPT
SMART-DATE (2018)	Post-PCI ACS patients	6-month DAPT	12-month DAPT	Death, MI, stroke	Similar MACCE but higher MI	Short DAPT not ideal in ACS
SMART-CHOICE (2019)	2993 PCI pts; ~58% non-ACS	3-month DAPT → P2Y12 monotherapy	12-month DAPT	Death, MI, stroke; BARC bleeding	Non-inferior ischemic outcomes, less bleeding	Mostly clopidogrel; multiple stents used
TWILIGHT-ACS (2020)	7119 high-risk PCI pts	3-month DAPT → ticagrelor monotherapy	Ticagrelor + aspirin	BARC 2–5 bleeding	Less bleeding, no ischemic harm	Supports aspirin withdrawal
TICO (2020)	3056 PCI pts with ACS (STEMI excluded)	3-month DAPT → ticagrelor monotherapy vs ticagrelor-based DAPT for 12 months	Composite of death, MI, stent thrombosis, stroke, target-vessel revascularization, or TIMI major bleeding	Primary endpoint reduced with ticagrelor monotherapy (driven by less bleeding)	MACCE similar; bleeding significantly lower	

GLOBAL LEADERS (2022)	15999 PCI pts (stable CAD & ACS ~47%)	1-month DAPT → ticagrelor monotherapy for 23 months vs standard DAPT	All-cause death or new Q-wave MI at 2 years	No significant difference in primary endpoint	Bleeding numerically lower but not significant
MASTER-DAPT (2021)	4579 HBR PCI pts	1-month DAPT vs ≥3-month DAPT	Net adverse clinical events and major bleeding	Non-inferior ischemic outcomes; significantly less bleeding	Supports very short DAPT in HBR patients
STOPDAPT-2 ACS (2022)	3005 PCI pts with ACS	1–2 month DAPT → clopidogrel vs 12-month DAPT	Death, MI, stroke, stent thrombosis, bleeding	Failed non-inferiority; more ischemic events	Clopidogrel monotherapy insufficient in ACS
STOPDAPT-3 (2024)	5966 PCI pts; 40% ACS, 60% stable CAD	No-DAPT strategy (prasugrel monotherapy) vs standard DAPT	Dual primary endpoints: bleeding and cardiovascular events	Bleeding non-inferior; ischemic events higher with no-DAPT	No-DAPT not suitable for broad PCI population
T-PASS (2024)	2853 PCI pts; ACS ~49%, STEMI included	1-month DAPT → ticagrelor monotherapy vs 12-month DAPT	Composite of CV death, MI, stroke, or BARC bleeding	Non-inferior ischemic outcomes; reduced bleeding	Supports short DAPT with potent P2Y12 inhibitor
ULTIMATE-DAPT (2024)	3453 ACS pts; guided by optical coherence tomography	1-month DAPT → ticagrelor monotherapy vs standard DAPT	BARC bleeding and MACCE	Significant bleeding reduction; no ischemic penalty	OCT-guided strategy improves safety

Shifting Paradigms: A Review of Recent Practice-Changing Cardiology Trials

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The landscape of cardiovascular medicine is shifting rapidly. From refining post-MI management to aggressive intervention in asymptomatic aortic stenosis, these studies demand a re-evaluation of our daily protocols. Here is a curated overview of the key evidence and its implications for our practice.

The Interventional Space: Precision and De-escalation

Perhaps the most immediately applicable findings come from the ischemic spectrum. We have long debated the necessity of beta-blockers in post-MI patients with preserved ejection fraction. The BETAMI-DANBLOCK trial provides a compelling answer. In patients with MI and LVEF >40%, beta-blocker therapy significantly reduced the risk of death or major adverse cardiovascular events (MACE) compared to no treatment (HR 0.85). Contrary to the trend of de-escalation, this suggests we should not be too quick to discard beta-blockade in this "low-risk" cohort (1).

Conversely, the push for de-escalating antiplatelet therapy hit a snag with the NEO-MINDSET trial. Attempting to switch to P2Y12 inhibitor monotherapy immediately after PCI for acute coronary syndromes (ACS) failed to show non-inferiority compared to standard DAPT regarding ischemic events. For now, premature DAPT discontinuation in the immediate post-ACS period remains risky (2).

However, procedural optimization continues to evolve. The IVUS-ACS trial reinforced the value of intravascular imaging, demonstrating that IVUS-guided PCI in diabetic patients with ACS significantly reduced target vessel failure by nearly half compared to angiography alone (3). This should encourage us to reach for the IVUS catheter more frequently in our high-risk diabetic patients.

On a practical note regarding patient comfort, the TONIC trial challenged the "NPO after midnight" rule. Allowing a light meal before non-urgent coronary procedures was non-inferior to fasting regarding safety endpoints (nausea, hypotension) and naturally improved patient satisfaction. This is a simple, patient-centered change we can implement immediately (4).

Complex PCI strategies were also refined. The PROCTOR trial addressed the dilemma of revascularization in post-CABG patients. It showed that targeting the native vessel yielded superior outcomes compared to treating the saphenous vein graft (SVG), driven largely by a reduction in periprocedural MI. Whenever technically feasible, we should prioritize the native vessel (5). Additionally, the SELUTION-DeNovo trial demonstrated that a strategy using Sirolimus-eluting balloons was non-inferior to drug-eluting stents (DES) for de novo lesions, potentially offering a "stent-free" future for select patients (6).

Heart Failure and Cardiomyopathy: Old Drugs and New Targets

In heart failure management, the DIGIT-HF trial revisited an old friend: digitoxin. In patients with advanced HFrEF (mean LVEF 29%), digitoxin significantly reduced the composite of death or HF hospitalization. Crucially, the benefit persisted even in patients on quadruple guideline-directed medical therapy (GDMT), with a safe therapeutic window. This revives a cost-effective option for our difficult-to-manage HFrEF patients (7).

However, not all novel therapies succeeded. The ODYSSEY-HCM trial investigated mavacamten, a game-changer for obstructive HCM, in non-obstructive symptomatic HCM. Unfortunately, it failed to show significant improvement in functional capacity or health status compared to placebo and was associated with more adverse events. For now, mavacamten remains strictly indicated for the obstructive phenotype (8).

Similarly, the CLEAR trial (using a factorial design for colchicine and spironolactone in MI) failed to show a benefit for routine colchicine use in reducing cardiovascular death or recurrent MI in this specific study design. This adds nuance to the inflammatory hypothesis, suggesting that colchicine may not be a universal "add-on" for all post-MI patients (9).

Structural Heart Disease: Earlier Intervention and Mechanical Support

Two trials stand out in the structural domain. The EARLY TAVR trial is arguably the most practice-changing. It demonstrated that in asymptomatic patients with severe aortic stenosis, early TAVR was superior to clinical surveillance in reducing death, stroke, or unplanned CV hospitalization. The "wait for symptoms" strategy appears outdated; we must now consider prophylactic intervention in these patients (10).

In the high-stakes arena of cardiogenic shock, the DANGER-SHOCK trial provided the first robust randomized evidence for microaxial flow pumps (Impella). Use of the device in STEMI-related shock significantly reduced 180-day mortality compared to standard care (45.8% vs. 58.5%). However, this survival benefit came at a cost of higher complication rates, including bleeding and limb ischemia. Careful patient selection and vascular access management are paramount (11).

Electrophysiology, PE, and Prevention

In electrophysiology, the OPTION trial explored combining atrial fibrillation ablation with Left Atrial Appendage (LAA) closure. This strategy was non-inferior to oral anticoagulation for efficacy but superior for safety, with significantly less non-procedure-related bleeding. This supports a "one-stop-shop" approach for select AF patients seeking to come off long-term anticoagulation (12).

For pulmonary embolism, the STORM-PE trial showed that computer-assisted vacuum thrombectomy (CAVT) was superior to anticoagulation alone in improving RV/LV ratios at 48 hours in intermediate-high risk PE. While mortality benefits require larger studies, the hemodynamic improvements are promising for acute management (13).

Finally, in lipid management, the VICTORION-Mono trial showed that Inclisiran monotherapy in primary prevention (patients without prior ASCVD) provided superior LDL-C reduction compared to ezetimibe or placebo. This opens the door for infrequent, injectable lipid-lowering therapy in lower-risk populations who may be statin-intolerant or prefer non-daily regimens (14).

Trial Name	Patient Population	Key Intervention	Primary Finding/Outcome
DIGIT-HF	HFrEF (LVEF < 40 %)	Digitoxin vs. Placebo	**Significant reduction** in death or HF hospitalization (39.5% vs. 44.1%, $p < 0.03$).
BETAMI-DANBLOCK	Post-MI with LVEF > 40%	Beta-blocker vs. No Beta-blocker	**Lower risk** of death or MACE in the beta-blocker group (14.2% vs. 16.3%, $p = 0.03$).
NEO-MINDSET	ACS after successful PCI	$P2Y_{12}$ inhibitor monotherapy vs. DAPT	Monotherapy was **not noninferior** for ischemic events but significantly reduced bleeding.

Trial Name	Patient Population	Key Intervention	Primary Finding/Outcome
TONIC	Nonurgent coronary procedures	Nonfasting vs. Fasting strategy	**Nonfasting was noninferior** in safety and improved patient comfort (less hunger/thirst).
IVUS-ACS	Diabetic patients with ACS	IVUS-guided PCI vs. Angiography-guided	**Reduced Target Vessel Failure (TVF)** at 1 year (3.6% vs. 8.3%, $p = 0.007$).
VICTORION-Mono	Low-risk, no prior ASCVD, LDL 100-190	Inclisiran monotherapy vs. Ezetimibe or Placebo	**Superior LDL-C reduction** with Inclisiran (46.5%) compared to ezetimibe or placebo.
DANGER-SHOCK	STEMI with cardiogenic shock	Microaxial flow pump vs. Standard care	**Lower 180-day mortality** with pump (45.8% vs. 58.5%), but higher adverse events.
ODYSSEY-HCM	Nonobstructive HCM	Mavacamten vs. Placebo	**No significant improvement** in functional capacity; benefits offset by LV dysfunction risk.
CLEAR	Post-MI (LVEF < 45 % or high risk)	Colchicine vs. Placebo	Colchicine **did not reduce** the incidence

Trial Name	Patient Population	Key Intervention	Primary Finding/Outcome
			of the composite primary outcome.
SELUTION-denovo	De novo coronary lesions	Drug-Eluting Balloon (DEB) vs. DES	**DEB was noninferior** to DES for Target Vessel Failure at one year.
STORM-PE	Int.-high risk Pulmonary Embolism	Mechanical Thrombectomy (CAVT) vs. Anticoagulation	**Superior reduction** in RV/LV ratio within 48 hours compared to anticoagulation alone.
OPTION	AFib after catheter ablation	LAA Closure vs. Oral Anticoagulation	**LAA closure was superior** in reducing bleeding and noninferior for efficacy.
PROCTOR	SVG stenosis requiring revascularization	Native vessel PCI vs. SVG PCI	**SVG PCI associated with better outcomes** (19% MACE) than native vessel PCI (34%).
EARLY TAVR	Asymptomatic severe aortic stenosis	Early TAVR vs. Clinical surveillance	**Early TAVR was superior** in reducing death, stroke, or unplanned hospitalizations.

Conclusion

These trials collectively push us toward more aggressive early intervention (EARLY TAVR), more precise procedural selection (IVUS-ACS, PROCTOR), and a nuanced use of pharmacotherapy (DIGIT-HF, BETAMI-DANBLOCK). While some new avenues closed (ODYSSEY for non-obstructive HCM), others have opened wide. As always, the art of cardiology lies in applying these statistical truths to the individual patient in front of us.

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Rythm versus Rate Control in AF - Has the Debate Settled

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Atrial fibrillation is the commonest form of sustained arrhythmia and has significant implications in clinical practice. It is associated with increase risk of stroke , HF , and higher mortality.

Early rhythm-control therapy — including a low threshold to offer catheter ablation in selected patients soon after diagnosis — reduces progression of AF, lowers important cardiovascular events in patients with recently diagnosed AF and comorbid cardiovascular disease, improves maintenance of sinus rhythm and quality of life, and may prevent AF-related remodeling that makes later treatment less effective.

Rationale for Early rhythm control:

• AF begets AF: electrical and structural remodeling (fibrosis, atrial dilatation) begin early; delaying effective rhythm control allows progression from paroxysmal to persistent AF, which is harder to eliminate and is associated with worse outcomes.

• Clinical impact of early intervention: the randomized EAST-AFNET 4 strategy (early rhythm control within 1 year of diagnosis) showed a significant reduction in the composite of cardiovascular death, stroke, HF hospitalization, or acute coronary syndrome versus usual care—supporting a disease-modifying effect beyond symptom relief.

Trial evidence supporting early rhythm control.

•**EAST-AFNET 4 (2020, NEJM)** — early rhythm-control (antiarrhythmics ± ablation as needed) reduced major CV outcomes vs usual care in patients with recently diagnosed AF and cardiovascular conditions.

•**EARLY-AF / CRYO-FIRST / STOP AF First trial** — first-line catheter ablation (cryoballoon or RF) outperformed antiarrhythmic drugs for freedom from recurrent atrial arrhythmia, improved quality of life, and—on longer follow-up—reduced progression to persistent AF. These trials support ablation as an effective initial rhythm-control strategy in appropriate patients.

The 2023 ACC/AHA/ACCP/HRS and 2024 ESC guidelines recommend early catheter ablation for AF in HFrEF (Class I/A) and for symptom and quality-of-life improvement in HFpEF.

Evidence in HFrEF: Landmark trials establish CA as disease-modifying therapy in AF with HFrEF. CASTLE-AF showed reduced mortality and HF hospitalizations with improved LVEF and quality of life in patients with LVEF ≤35%.AMICA showed no benefit in more advanced HF, likely reflecting irreversible disease. CAMERA-MRI demonstrated marked LVEF recovery and fibrosis regression in AF-mediated cardiomyopathy, while AATAC showed CA was superior to amiodarone for rhythm maintenance, mortality, and HF hospitalization. Meta-analyses confirm reductions in death, HF admissions, and improved LV function versus medical therapy. Benefits extend into advanced HF (CASTLE-HTx) and across the EF spectrum (RAFT-AF, CABANA subanalyses), though mortality benefit is strongest in HFrEF.

Recent major society guidance has moved toward earlier consideration of rhythm control and recognizes catheter ablation as an effective option for achieving rhythm control and for improving outcomes in selected patients (see recent updates from the European Society of Cardiology and the American College of Cardiology). These documents emphasize individualized decisions: symptom burden, type of AF, comorbidities (especially heart failure), and patient preference.

Practical argument for early catheter ablation (concise points for a cardiology audience)

1. **Higher single-procedure efficacy if AF is recent** — less substrate complexity better success and less progression to persistent AF.
2. **Reduces AF recurrence & progression** — randomized first-line ablation trials show superior rhythm outcomes vs antiarrhythmics, and longer follow-up shows less progression to persistent AF
3. **Symptom and QoL gain, potential downstream outcome benefit** — early rhythm control reduced composite CV events in EAST-AFNET 4; while ablation trials vary for hard endpoints, subgroup data (and HF trials) show meaningful clinical benefit.
4. **Safety profile acceptable in contemporary practice** — procedural safety has improved; risks must be weighed against ongoing AF morbidity and risks of antiarrhythmic therapy.
5. **Patient selection maximizes value** — ideal candidates: recent-onset symptomatic paroxysmal or early persistent AF, younger patients with fewer comorbidities, those intolerant/failing antiarrhythmics, and patients with HF where ablation may improve LVEF.

Offer rhythm control early in the AF course; consider catheter ablation as a first-line or early option for eligible patients because timely restoration of sinus rhythm preserves atrial substrate, improves symptoms/QoL, reduces AF progression, and—when delivered as part of an early-rhythm strategy—can lower major cardiovascular events.

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Tailoring Guideline Directed Medical Therapy (GDMT) after STEMI- Current Evidence

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Management of ST segment elevation myocardial infarction (STEMI) has evolved from the days of masterly inactivity to aspirin and beta blockers, through fibrinolysis and combined antithrombotics, statins, ACE inhibitors and culminating into primary angioplasty. With this aggressive management, the post MI left ventricular dysfunction and recurrent ischemic events have drastically reduced, along with a dramatic reduction of mortality.

However, the onus has now been shifted to post hospital discharge and long term management after STEMI. Unfortunately most trials with most medicines have limited duration of follow up, typically around 3 years on an average. So, whether some medicines need to be continued indefinitely is determined more by wisdom and practice, rather than backed up by solid evidence.

Let us focus here on the post discharge and short term continuation of different first line medicines.

Dual antiplatelet therapy (DAPT) is class I recommendation by all guidelines. Aspirin is the common denominator, the second agent being clopidogrel, ticagrelor or prasugrel. Typically with primary PCI, ticagrelor is the drug of choice. In some countries like Germany, prasugrel is preferred, especially in the diabetic subjects. Patients managed medically or with only fibrinolysis are still commonly on clopidogrel. When the thrombotic risk and bleeding risk are balanced, the DAPT typically continues for 6 months; being curtailed to 3 months if bleeding risk is high, and being extended to 12 months if the thrombotic risk is high. After 12 months, in a stable patient, the options are wide open, from SAPT with aspirin or clopidogrel or ticagrelor to DAPT with aspirin and clopidogrel to DAT with aspirin and low dose rivaroxaban. And this can go on for 3-7 years, and beyond as a data free zone. In presence of atrial fibrillation, when a full dose of DOAC comes by default, the DAPT is typically aspirin and clopidogrel, for 7 days to 1 month to 3 months depending again on individual preference and bleeding and thrombotic risk weightage; followed by DOAC with clopidogrel till 12 months and then DOAC alone.

Statins are the next most important agents for prognostic benefit. Dyslipidemia guidelines by the Cardiological Society of India recommend loading in the emergency room with 40 mg rosuvastatin or 80 mg atorvastatin along with 10 mg ezetimibe in all patients irrespective of their lipid values or background treatment except for elderly above 75 years and with CKD stages 3 to 5, where the statin is given in half the dose. After 4 weeks, if target LDL value is not achieved, bempedoic acid is added. In another 4 weeks if the target is still elusive, then evolocumab or inclisiran injection is to be recommended. The optimum regimen should continue for indefinite period as the secondary prevention of MACE primarily depends on LDL level.

Rennin-angiotensin-aldosterone system blockers (RAASI) are essential post-MI medicines for prognostic benefits. ACEI is the drug of choice, typically starting as soon as the patient is hemodynamically stable, and especially in anterior wall MI and MI with LV dysfunction. In ACEI intolerant patients, ARBs like valsartan or losartan should be used. The new RAASI, sacubitril-valsartan, has failed to show incremental benefit over ACEI. However, after 3 months, if the patient is in HFrEF or HFmrEF, then there is a strong case to replace ACEI with sacubitril-valsartan.

Lastly, the beta blockers are extremely useful in acute STEMI to prevent sudden cardiac death, though in hemodynamically unstable patients, they may cause acute HF or cardiac rupture. In chronic situation if the patient is in HFrEF, then beta blockers should continue indefinitely. But, otherwise their long term use is debatable. Usually in hypertensive patients after MI, beta blockers offer survival advantage over other anti-hypertensives for up to 2 years, beyond which their usefulness is questionable. In non-hypertensive subjects, their use after hospital discharge has been fraught with recent conflicting evidences, unless there is another clear indication for their use.

Vaccinations against influenza, pneumonia and herpes zoster have been recommended with variable emphasis by different guidelines, and largely guided by co-morbid conditions like HF, COPD, etc.

Use of anti diabetics is influenced after STEMI, where SGLT2I and GLP1A are considered first line.

Life style measures including diet, exercise, weight management, abstinence from tobacco and limiting alcohol consumption, yoga and anxiety management, etc, are also essential guideline recommendations after STEMI. `

Here I tried to give a simplistic overview of post-STEMI management and deliberately avoided mention of trials and references. I am sure that the interested and initiated doctors will explore the relevant sections themselves.

When to Refer a Patient of Acyanotic CHD for Catheter Intervention / Surgery

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When to Refer a Patient of Acyanotic CHD for Catheter Intervention / Surgery

Introduction to Acyanotic Congenital Heart Disease (CHD)
Acyanotic CHD refers to a group of congenital heart defects where there is no mixing of oxygenated and deoxygenated blood leading to cyanosis at rest. These defects typically involve left-to-right shunts or obstructive lesions, resulting in volume or pressure overload on the heart. Common acyanotic CHDs include atrial septal defect (ASD), ventricular septal defect (VSD), patent ductus arteriosus (PDA), coarctation of the aorta (CoA), aortic stenosis (AS), pulmonic stenosis (PS), and atrioventricular septal defect (AVSD). The decision to refer a patient for catheter intervention or surgery depends on factors such as defect size, hemodynamic impact, symptoms, age, pulmonary vascular resistance, and associated complications like heart failure, pulmonary hypertension, or arrhythmias. Guidelines from organizations like the American College of Cardiology (ACC) and American Heart Association (AHA) emphasize timely intervention to prevent irreversible damage, while favouring minimally invasive catheter-based approaches when feasible.

Referral is generally indicated when the defect causes significant shunt (e.g., Qp:Qs ratio >1.5:1), chamber enlargement, elevated pressures, or clinical symptoms like failure to thrive, recurrent infections, or exercise intolerance. In adults, additional considerations include pulmonary arterial hypertension (PAH) and comorbidities. Catheter interventions, such as device closure for shunts or balloon valvuloplasty for stenoses, are preferred for their lower morbidity, but surgery is reserved for complex cases or failures of percutaneous methods.

Atrial Septal Defect (ASD)
ASD involves an opening in the atrial septum, leading to left-to-right shunting and right ventricular (RV) volume overload. Indications for referral include evidence of RV enlargement on echocardiography, paradoxical embolism, or symptoms like dyspnoea or atrial arrhythmias. Closure is recommended for secundum ASDs with Qp:Qs >1.5:1 and no severe PAH (pulmonary vascular resistance <5 Wood units). In adults, unrepaired ASD with PAH requires risk assessment by specialists before intervention.

Timing: Asymptomatic children are referred between 2-4 years of age to allow spontaneous closure while preventing long-term complications. Symptomatic infants or those with failure to thrive warrant earlier referral, often by 1 year. In adults, referral is prompted by progressive RV dilation or new symptoms. Catheter-based closure using devices like Amplatzer is first-line for secundum ASDs, with surgery for primum or sinus venosus types or large defects (>38 mm). Contraindications include Eisenmenger syndrome, where closure could worsen outcomes.

Ventricular Septal Defect (VSD)
VSD is a hole in the ventricular septum, causing left-to-right shunting. Small VSDs (<3 mm) often close spontaneously and require monitoring only. Referral for intervention is indicated for moderate-to-large VSDs with Qp:Qs >1.5:1, left ventricular (LV) volume overload, PAH, aortic regurgitation due to cusp prolapse, or symptoms like congestive heart failure (CHF) and poor growth.

Timing: Small asymptomatic VSDs without PAH or LV dilation are observed until adulthood. For large VSDs with controlled CHF, refer by 3-6 months; uncontrolled symptoms necessitate immediate referral. VSD with aortic cusp prolapse and more than trivial regurgitation requires prompt intervention to prevent progression. In adults, unrepaired VSD with PAH needs specialist evaluation; closure is considered if there's LV enlargement or endocarditis risk. Catheter closure is suitable for muscular VSDs, while peri membranous types often require surgery due to conduction risks.

Patent Ductus Arteriosus (PDA)
PDA is persistence of the fetal ductus arteriosus, leading to shunting. Silent small PDAs may not require intervention, but audible one's risk endocarditis. Referral is indicated for moderate-to-large PDAs with LV overload, PAH, or symptoms like CHF.

Timing: Small audible PDAs are closed after 1 year. Large PDAs with controlled symptoms are referred by 3-6 months; neonates with CHF need urgent closure. In adults, intervention is for LV dilation or PAH, but contraindicated in severe PAH. Catheter coil or device occlusion is preferred; surgery for very large or aneurysmal PDAs.

Coarctation of the Aorta (CoA)
CoA is narrowing of the aorta, causing upper body hypertension and LV pressure overload. Indications include gradient >20 mmHg, hypertension, or CHF. Associated bicuspid aortic valve or aneurysms warrant monitoring.

Timing: Neonatal critical CoA with ductal dependence requires immediate prostaglandin and intervention. Older children/adults with hypertension or gradient are referred electively. Balloon angioplasty with stenting is common in older patients; surgery for infants or complex anatomy.

Aortic Stenosis (AS)
AS involves LV outflow obstruction. Critical AS in neonates presents with shock and requires urgent balloon valvuloplasty. Mild AS (gradient <40 mmHg) is monitored; moderate-severe (gradient >40 mmHg) with symptoms or LV hypertrophy indicates referral.

Timing: Asymptomatic mild cases followed annually. Symptomatic or severe AS (gradient >64 mmHg) needs prompt intervention. Catheter valvuloplasty first-line; surgery for sub/supra-valvar or failed procedures.

Pulmonic Stenosis (PS)
PS causes RV outflow obstruction. Indications: Gradient >40 mmHg, RV hypertrophy, or symptoms. Critical PS in neonates needs urgent intervention.

Timing: Mild PS observed. Moderate-severe referred electively in childhood. Balloon valvuloplasty is standard; surgery rare.

Atrioventricular Septal Defect (AVSD)
AVSD combines ASD, VSD, and AV valve issues. Partial AVSD behaves like ASD; complete like large VSD. Referral for large shunts, AV regurgitation, or CHF. In adults, cardiac catheterization assesses PAH.
Timing: Partial: 2-4 years if asymptomatic. Complete: 3-6 months if controlled. Surgery is primary; catheter rare.

General Considerations
Referral decisions integrate echocardiography, clinical status, and sometimes catheterization for PAH assessment. Early intervention prevents PAH progression, but delays in asymptomatic cases allow growth. In adults, focus on quality of life and comorbidities. Multidisciplinary teams at specialized centres optimize outcomes.

ACS and LV dysfunction: where do ARNI and SGLT-2i therapy stand?

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LV dysfunction is very common in the post-ACS setting- incidence of upto 60% (based on the different definitions used). It is a strong predictor of mortality. According to a study, death or hospitalization for heart failure was observed in 26.9-37.6% of patients with post-MI LV dysfunction at 3 years. Despite revascularization in the acute phase, LV dysfunction may progress in many patients due to adverse LV remodelling. It is thus important to start GDMT to prevent worsening in these patients- includes beta blockers, ACEi/ARB, MRA. There is evolving evidence on the role of ARNI and SGLT-2i in this subset of patients despite proven beneficial effect in the chronic HFrEF group.

Sacubitril/Valsartan (ARNI) blocks the AT1 receptor of angiotensin and inhibits the RAAS pathway, thereby reducing BP, beneficial effect on LV remodelling. On the other hand, it inhibits neprilysin which degrades the natriuretic peptides, causing natriuresis and beneficial effect on the heart. In the post-MI setting, there is adverse LV remodelling due to activation of the sympathetic system, RAAS activation. Hence, it is believed that ARNI will have beneficial effects in this group of patients when started early. The PARADISE-MI trial was conducted to address this question. It was a multicentric, international, randomised, active controlled, double blind, phase III trial comparing the safety and efficacy of sacubitril/valsartan versus ramipril in preventing heart failure and CV death after high risk AMI. The study had 5669 participants. It included patients in the initial 7 days of post-ACS with LVEF<40% or evidence of pulmonary congestion, with risk augmenting factors. The study did not show any significant decline in primary endpoint compared to ramipril. It showed trends towards benefit in the sacubitril/valsartan group but these were not statistically significant. Latest Guidelines recommend using ACE-/ARB in the early post-MI phase with switching to ARNI if persistent LV dysfunction develops, after stabilization weeks to month later.

Similar to ARNI, there is a great amount of evidence supporting the beneficial effect of SGLT-2i therapy in chronic HFrEF group of patients. SGLT-2i unload the heart by diuresis, improves cardio metabolic profile, and reduces the adverse remodelling of the heart. Its use is thus believed to have beneficial effect in the post-MI setting. EMMY trial was the first proof of concept trial which proved the safety of SGLT-2i in the post-MI setting. However, the trial was not powered to measure hard CV outcomes (death, HF hospitalization). EMPACT-MI trial evaluated the role of Empagliflozin in the early post-MI phase. It recruited 6522 patients with initiation of Empagliflozin in the initial 14 days post-MI, followed up to 18 months. Empagliflozin did not reduce the composite of HF hospitalization and CV death, nor was any benefit seen in the all-cause mortality or CV mortality. But, there was significant reduction in first HF hospitalization and total HF hospitalization. Another large trial- the DAPA MI trial evaluated the role of SGLT-2i in the post-ACS setting. It did not show any benefit in terms of mortality or HF hospitalization, but showed better cardio metabolic profile, reduced incidence of new onset DM and weight reduction. Till date, guidelines do not give a specific class of recommendation to the SGLT-2i but promote the early use in post-ACS setting once the patient is stable.

CABG VS PCI For Left Main Disease

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The treatment of left main (LM) coronary artery disease (CAD) requires complex decision-making. Patients with untreated significant left main coronary artery (LMCA) disease have a poor prognosis The natural history of medically treated left main (LM) disease has been associated with 73% mortality at 15 years, and revascularization with coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI) is indicated.

There is continued debate on the optimal revascularization strategy for patients with left main disease. Recent clinical practice guidelines provide clinicians with guidance; however, decisions regarding treatment for individual patients can still be difficult

The evidence from randomized clinical trials (RCTs) and meta-analyses, suggest equivalent results for the composite of death, myocardial infarction (MI), and stroke up to 5 years of follow-up when comparing CABG with PCI but there is increased risk of repeat revascularization in patients who had undergone PCI.

However, most completed trials in left main disease may have limited relevance to contemporary practice because of technological and pharmacological advances that have changed the practice of PCI, including the introduction of safer and more effective drug-eluting stents; more judicious use of PCI, based on fractional flow reserve measurements; PCI optimization with intravascular ultrasound and optimal coherence tomography; and improved antithrombotic and antiplatelet agents.

In addition, the use of risk stratification and assessment tools, such as the baseline and residual SYNTAX scores, which assess the potential extent of revascularization, may be helpful in identifying patients who may have comparable or superior outcomes with PCI compared with CABG.

CABG has also evolved with greater use of multiple arterial grafts, although it is not yet certain that this has an impact on mortality. Moreover, substantial improvements in secondary prevention and guideline-directed medical therapy have improved outcomes for all patients with ischemic heart disease.

The recommendations for choosing mode of revascularization of unprotected LMCA disease is based on coronary anatomical complexity assessed by the Synergy between PCI with Taxus and Cardiac Surgery (SYNTAX) score, individual cardiac and extracardiac characteristics, and patient preference.

When weighing the risks and benefits of PCI vs CABG for LM disease, it is important to take into account the factors that are important to the patient first and foremost, while also considering clinical factors that may affect optimal outcomes.

For patients with a low SYNTAX score (≤22), PCI and CABG have the same class and level of recommendation (I A), but for patients with intermediate SYNTAX score (23–32) the recommendation for CABG is I A and for PCI IIa A. Only CABG is recommended (I A) for patients with very complex coronary anatomy (SYNTAX score ≥33) and PCI is not recommended (III B) for such patients.

Factors that may favor CABG include diabetes mellitus (especially with concurrent significant MVD), a contraindication to dual antiplatelet therapy, history of recurrent in-stent restenosis, or concomitant ascending aortic or valvular pathology with an independent indication for surgery.

Clinical factors that may favor PCI include clinical frailty or severe comorbidities that may affect a patient's ability to rehabilitate after CABG. Some of these comorbidities are severe chronic obstructive pulmonary disease, severe chest deformity, prior sternotomy and/or lack of conduits, sequelae of prior chest radiation, advanced chronic kidney disease, immunosuppression, reduced life expectancy, or suboptimal psychosocial support.

Integrating a multidisciplinary heart team into institutional practice provides a formalized approach to evaluate complex LM disease and to best ensure standardized and equitable care delivery. The decision regarding revascularization with either PCI or CABG should be made by a heart team after considering each patient's individual circumstances, including life expectancy, comorbidities, extent of disease, angiographic anatomy, likelihood and perceived need for complete revascularization, and patient preference.

Evaluation and Management of Chest Pain with Non-obstructive CAD (ANOCA/INOCA/MINOCA)

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At the outset, I find the term of chronic stable angina syndrome as misleading as it is neither chronic in subsets nor limited to angina, as many times as some subsets are unstable and shift to the zone of NSTEMI with rise of troponins. The designation of chronic stable angina leads to passivity in minds of treating cardiologist that it is a passive phenomenon which doesn't require our utmost attention like the situations of acute coronary syndromes, which may not be true often.

Chronic stable angina[{CSA] is a heterogenous entity with many active zones ,so we need to change our strategy to have a fresh look towards CSA.

One such new concept is of ANOCA,INOCA and MINOCA with angina, ischemia and myocardial infarction in presence of non obstructive coronary artery disease. These categories are not benign and have poor prognosis as compared to patients with normal coronaries. Longterm it leads to future cardiovascular events and considerable disability. In group of CSA for elective angiography, ANOCA accounts for 40 to 70 percent cases while INOCA FPR 20 TO 30 PERCENT OF THEM..MINOCA should angiographically show lesions not exceeding 50 percent of the vessel diameter. Three different types of plaque disruption are plaque rupture, erosion or calcified nodule, which can only be detected by intravascular imaging and its overall prevalence is 6 to 8 percent.

We should differentiate these from similar conditions like myocarditis, takasubo cardiomyopathies or spontaneous coronary artery dissection. Imaging like cardiac MRI can differentiate myocarditis from ACS on the basis of late gadolinium enhancement patterns.

To understand pathophysiology of MINOCA, we need to look at epicardial level for plaque erosion or disruption ,rule vasospasm and also evaluate the factor of vasospasm. Another challenge is to look for microvascular causes and coronary microvascular dysfunction.

Although management is not grossly affected but non invasive techniques like stress echocardiography, PET Scan, CMR, CCTA, Calculation of FFR or iFR and miscellaneous tests like ergonovine and other vasodilator tests help in better evaluation of MINOCA. The role of coronary flow reserve is equally important to evaluate microvascular dysfunction. subset. Definitely IVUS and OCT can help us to significant extent in understanding plaque features and the mechanisms for ischemia

The three major cornerstones of management are lifestyle changes, control of CV risk factors and pharmacological therapy. Apart from beta or calcium blockers, aCE inhibitors, statins, nitrates and antiplatelets are also used commonly. Nikorandil has special place to tackle microvascular dysfunction, as it relaxes smooth muscles and leads to synhesis of NO. Second line drugs like Ranolazine, trimetazidine and Ivabradine can be better utilised for vanagement of angina or ischemia.. Some newer drugs are endothelin antagonists like zibotentan, Rho kinase inhibitors like fasudil and xanthine derivatives to release adenosine during exercise. Finally the role of coronary sinus reduction device in treatment of microvascular

dysfunction is to be seen ANOCA, INOCA and MINOCA are complex to understand as severity of ischemia doesn't match with level of atherosclerosis. If left untreated, there is a fair chance of serious cardiovascular events in future.

Multidisciplinary approach and personalised treatment is essential for better management.

Latest Hypertension Guidelines - The Key Takeaways

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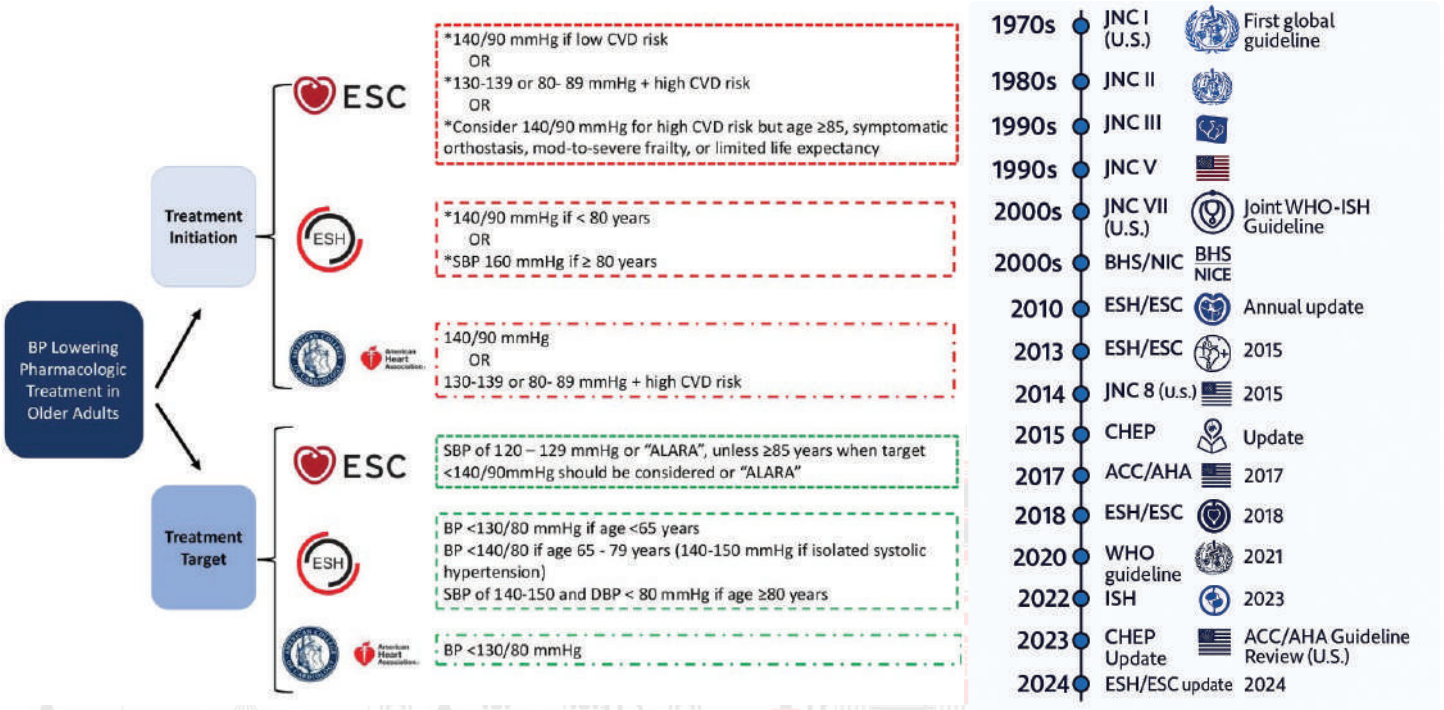
Hypertension guidelines released between 2020 and 2025 paint a stark picture: over 1.4 billion adults worldwide live with high blood pressure, yet fewer than one in five achieve adequate control in most countries, and India mirrors this gap with nearly 60% of treated patients remaining uncontrolled. The updated international recommendations now converge on earlier diagnosis, tighter blood pressure targets, and routine use of combination therapy—especially single pill combinations—to prevent cardiovascular, renal, and brain complications across the life course.

Globally, four major frameworks now drive practice: the International Society of Hypertension 2020, European Society of Hypertension 2023, European Society of Cardiology 2024, and the 2025 American Heart Association/American College of Cardiology guideline. The 2025 AHA/ACC document retains a clinic threshold of at least 130/80 mmHg for defining hypertension in most adults, with categories of normal, elevated, stage 1, and stage 2 guiding intensity of follow up and therapy. A risk stratified approach is central: pharmacologic treatment is initiated earlier (from 130/80 mmHg) in people with established cardiovascular disease, diabetes, chronic kidney disease, target organ damage, or high 10 year risk, while lifestyle measures are recommended at every level of blood pressure.

The 2024 ESC guideline reinforces a similar risk based strategy, recommending prompt initiation of drug therapy when blood pressure is at least 140/90 mmHg regardless of risk, and even earlier treatment thresholds for those with moderate or severe CKD, documented CVD, hypertension mediated organ damage, diabetes, or familial hypercholesterolaemia. Both European and American documents highlight the persistent demographic gaps in awareness, treatment, and control—particularly among younger adults, men, and Asian populations—underlining the need for targeted implementation efforts.

On treatment, the slides emphasize that most patients will not reach targets on monotherapy, making combination therapy the default rather than the exception. First line drug classes include ACE inhibitors or ARBs, calcium channel blockers, and thiazide/thiazide like diuretics, with beta blockers reserved for compelling indications such as heart failure, post MI, angina, or atrial fibrillation. Single pill combinations are promoted to simplify regimens, improve adherence, and accelerate attainment of target blood pressure, while structured algorithms address resistant hypertension, from ruling out pseudoresistance to optimizing multi drug therapy and referring to specialists.

Non pharmacologic therapy remains the foundation of care, with the DASH diet, sodium restriction, and modest weight loss each providing clinically meaningful reductions in systolic blood pressure—up to about 11 mmHg in people with hypertension. The presentation closes by stressing that despite robust evidence and clear algorithms, control rates are unacceptably low, particularly in low and middle income countries, making health system strengthening, home BP monitoring, and rapid treatment intensification essential to translate guidelines into fewer strokes, myocardial infarctions, kidney failure, and cases of dementia.



Exploring the Therapeutic Landscape in Hypertrophic Cardiomyopathy : Current Insights

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Hypertrophic cardiomyopathy (HCM) represents a primary myocardial disorder characterized by left ventricular hypertrophy, often leading to dynamic obstruction and impaired cardiac function. This synopsis draws insights from the recent evidence to outline key therapeutic considerations for cardiologists managing symptomatic patients.

Background

HCM manifests as a chronic, progressive condition driven by sarcomeric dysfunction, resulting in excess myosin-actin cross-bridging, hypercontractility, and impaired myocardial relaxation. Patients commonly experience dynamic left ventricular outflow tract (LVOT) obstruction, particularly in the obstructive subtype, which predominates among cases and contributes to exertional dyspnea, chest pain, palpitations, and presyncope. Diagnosis relies on comprehensive imaging, including echocardiography with provocative maneuvers like Valsalva or exercise to unmask latent gradients, alongside family history and electrocardiographic findings such as repolarization abnormalities.

Early recognition through targeted physical exam findings, like a systolic murmur diminishing with squatting, prompts essential echocardiographic assessment to differentiate obstructive from non-obstructive forms and guide risk stratification. Disease progression often involves fibrosis, atrial fibrillation, and heart failure symptoms, underscoring the need for vigilant monitoring to preserve functionality and quality of life.

Gaps in Treatment

Standard-of-care therapies, including beta-blockers and non-dihydropyridine calcium channel blockers, provide symptomatic relief by reducing heart rate and contractility but fail to address underlying sarcomeric pathology. These agents often yield incomplete LVOT gradient reduction, leaving many patients with persistent exertional limitations and high symptomatic burden despite maximal titration. Disopyramide offers additive benefit in select cases but introduces challenges with tolerability and lacks disease-modifying effects.

A substantial proportion of patients remain refractory to pharmacotherapy, necessitating invasive septal reduction therapies (SRT) like myectomy or alcohol septal ablation for those with severe symptoms and qualifying gradients; however, these carry procedural risks and do not halt intrinsic myocardial remodelling. This highlights the unmet need for targeted interventions that improve compliance, reduce wall stress, and enhance exercise capacity without relying solely on non-specific symptom palliation.

Evidence behind Mavacamten

Mavacamten, a cardiac myosin inhibitor, directly mitigates excess cross-bridge formation, alleviating hypercontractility and LVOT obstruction in symptomatic obstructive HCM. In the EXPLORER-HCM trial, patients on background therapy showed meaningful improvements in exercise capacity, NYHA class, and post-exercise gradients, with benefits extending into long-term extensions demonstrating sustained echocardiographic and biomarker responses. The VALOR-HCM study further evidenced its utility in severely symptomatic, SRT-eligible patients, markedly reducing guideline eligibility for invasive procedures while enhancing functional status.

Echo-guided dose titration enables individualized therapy, yielding early symptom relief, reduced NT-proBNP levels reflecting lower wall stress, and high completion rates; transient LVEF reductions remain manageable with monitoring and interruption protocols. Long-term data affirm tolerability, positioning mavacamten as a bridge or alternative to SRT, with key benefits in patient-reported outcomes like shortness of breath and health status.

Guidelines

Recent AHA/ACC guidelines endorse mavacamten for adults with NYHA class II-III obstructive HCM who remain symptomatic despite beta-blockers or calcium channel blockers, emphasizing shared decision-making with serial echocardiography. ESC recommendations align, advocating myosin inhibitors post-first-line therapy failure, prior to SRT consideration in eligible patients with persistent gradients and symptoms. Risk assessment for sudden death, atrial fibrillation management, and lifestyle modifications complement pharmacotherapy across both frameworks.

Initiate mavacamten at low doses with LVEF and gradient monitoring every 4-12 weeks to optimize response; contraindicate in those with LVEF below threshold or recent SRT. Guidelines stress multidisciplinary care, integrating genetic counselling and comorbidity control to mitigate progression and complications.

Vulnerable Plaques - Identification and Management

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Vulnerable plaque refers to high-risk atherosclerotic lesions prone to rupture, leading to acute coronary syndromes like myocardial infarction. These plaques are characterized by a thin fibrous cap, large lipid-rich necrotic core, inflammation, and positive remodeling, distinguishing them from stable plaques.[1][4]

Pathophysiology

Vulnerable plaques, often termed thin-cap fibroatheromas (TCFA), feature a fibrous cap less than 65 micrometers thick overlying a necrotic core exceeding 30% of plaque volume. Macrophage infiltration drives cap thinning via matrix metalloproteinases, while neovascularization and intraplaque hemorrhage exacerbate instability. Rupture exposes thrombogenic contents, triggering thrombosis in about two-thirds of acute events; erosion without rupture accounts for the rest.[3][4][1]

Diagnostic Methods

Intracoronary imaging excels in vulnerable plaque detection during catheterization. Optical coherence tomography (OCT) offers 10-20 micrometer resolution to precisely measure cap thickness, identify microchannels, and detect macrophages. Intravascular ultrasound (IVUS), including virtual histology IVUS (VH-IVUS), assesses plaque burden, composition (fibrous, fibro-fatty, necrotic, dense calcium), and minimum lumen area. Near-infrared spectroscopy (NIRS) quantifies lipid content via chemograms, with maximal lipid-core burden index predicting events. Combined NIRS-IVUS identifies high-risk features synergistically.[5][1]

Non-invasive options lag but advance rapidly. Coronary computed tomography angiography (CCTA) spots low-attenuation plaques (<30 HU), napkin-ring signs, and positive remodeling; its prognostic value grows with AI integration. Vessel wall high-resolution MRI evaluates cap status, hemorrhage, and inflammation, correlating well with histopathology. Positron emission tomography targets inflammation via 18F-FDG uptake. These guide risk stratification but lack routine adoption pending prophylactic therapy validation.[7][3]

Management Strategies

Aggressive medical therapy stabilizes plaques primarily. High-intensity statins (e.g., atorvastatin 40-80 mg) thicken caps, shrink necrotic cores, and reduce events by 20-30% in trials like ASTEROID and REVERSAL. PCSK9 inhibitors (evolocumab, alirocumab) further regress atheroma volume. Anti-inflammatories like colchicine (COLCOT, LoDoCo2 trials) cut events by targeting residual inflammatory risk. Dual antiplatelet therapy post-event prevents thrombosis, while lifestyle changes (smoking cessation, diet, exercise) are foundational.[10][1][3]

Interventional approaches target plaques proactively. Preventive percutaneous coronary intervention (PCI) stents non-obstructive (<50-70% stenosis) high-risk lesions identified by imaging. Drug-eluting stents (DES) increase cap thickness and minimum lumen area; bioresorbable scaffolds restore vasomotion while passivating plaques. Trials like PROSPECT ABSORB and FIRE show safety and stabilization, reducing major adverse cardiac events. Drug-coated balloons deliver antiproliferative agents without permanent implants, under investigation (e.g., BASKET-SMALL 2). PREVENT trial (2024 data) supports imaging-guided PCI for thin-cap fibroatheromas.[4][9][3]

Challenges and Future Directions

Prospective randomized trials (e.g., PREVENT, ILUMIEN IV) affirm imaging-directed strategies reduce events versus angiography alone, but cost, expertise, and lesion multiplicity limit uptake. Non-invasive tools must evolve for screening; AI-enhanced CCTA shows promise. Hybrid therapies combining pharmacology, preventive PCI, and novel agents (e.g., anti-IL-6) may personalize care. Ongoing studies prioritize clinical endpoints over surrogates like plaque volume.[2][3][4]

Overall, vulnerable plaque management shifts from reactive to proactive, leveraging multimodal imaging and targeted interventions for superior outcomes. (Word count: 548)[1][5]

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Optimising Outcomes for Hypertension in Uncontrolled BP and CAD Patients

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Hypertension Management in CAD Patients Hypertension is a major modifiable risk factor for the development and progression of coronary artery disease (CAD). Effective blood pressure (BP) control in patients with CAD significantly reduces the risk of myocardial infarction, stroke, heart failure, and cardiovascular mortality. Contemporary guidelines emphasize individualized BP targets, careful drug selection, and avoidance of excessive BP lowering, particularly of diastolic pressure. Current evidence supports initiation of antihypertensive therapy in CAD patients with BP ≥130/80 mmHg, with a target BP of <130/80 mmHg when tolerated. European guidelines further recommend achieving a systolic BP of 120–129 mmHg in most patients, while cautioning against diastolic BP <60 mmHg due to the risk of compromised coronary perfusion and ischemia, especially in elderly patients. Lifestyle modification forms the cornerstone of management and includes dietary sodium restriction, weight control, regular physical activity, smoking cessation, moderation of alcohol intake, and stress management. These measures complement pharmacotherapy and contribute to overall cardiovascular risk reduction. Pharmacologic therapy should be tailored to both BP control and ischemic benefit. ACE inhibitors or ARBs are foundational agents due to their proven cardiovascular protection. Beta-blockers are particularly beneficial in patients with prior myocardial infarction, angina, or left ventricular dysfunction. Calcium channel blockers and thiazide or thiazide-like diuretics are effective adjuncts, often used in combination therapy to achieve BP targets. Single-pill combinations are encouraged to improve adherence. Special consideration is required in elderly and frail patients, where less aggressive BP targets may be appropriate. Continuous monitoring is essential to balance optimal BP control against the risk of hypotension and myocardial ischemia. In summary, hypertension management in CAD requires a patient-centered approach integrating guideline-directed BP targets, lifestyle intervention, and judicious pharmacologic therapy to improve long-term cardiovascular outcomes.

GLP-1 Analogue Based therapies: Which One to Choose for Cardiovascular Disease Patients?

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Introduction

Obesity, Cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM) cluster together to increase morbidity and mortality [1]. Incretin-based therapies have evolved from glycemic agents to powerful cardio-metabolic drugs with proven benefits beyond glucose control. GLP-1 receptor agonists (GLP-1 RAs) have shown robust cardiovascular outcome trial (CVOT) evidence, while dual GLP-1/GIP and triple GLP-1/GIP/glucagon agonists represent next-generation therapies with increased efficacy [2]. This review compares mechanisms, efficacy, and cardiovascular evidence to guide therapy selection in CVD patients.

Mechanisms of Action

GLP-1 Receptor Agonists mimic endogenous GLP-1, enhancing glucose-dependent insulin secretion, suppressing glucagon, delaying gastric emptying, and reducing appetite[1]. GLP-1 RAs cause weight loss (8–15%), reduce visceral adiposity, improve blood pressure and lipids, and exert anti-inflammatory and endothelial protective effects with significant cardiovascular benefits demonstrated in landmark trials including LEADER (liraglutide), SUSTAIN-6 (semaglutide), REWIND (dulaglutide), and HARMONY Outcomes (albiglutide)[3].

Dual GLP-1/GIP Agonists (e.g., tirzepatide) add GIP receptor activation, further augmenting insulin secretion and modulating adipose metabolism[2]. Across the SURPASS phase 3 program (including SURPASS-2, SURPASS-3, and SURPASS-5), this dual mechanism yields greater appetite suppression, superior weight loss (15–20%), more HbA1c reductions (2.0–2.5% vs 1.0–1.8% for GLP-1 alone), and greater improvements in insulin sensitivity and lipid parameters while maintaining low hypoglycemia risk[1][2].

Triple GLP-1/GIP/Glucagon Agonists (e.g., retatrutide) has additional glucagon receptor agonism, increasing energy expenditure and fat oxidation[4]. In a phase 2 trial of retatrutide in obesity (published in NEJM, June 2024), mean weight loss reached approximately 24.2% at 48 weeks, the greatest reported pharmacological weight reduction, alongside strong improvements in glycemia, liver fat, and metabolic risk markers [4]. However, long-term cardiovascular and renal outcome data is still to come [4].

Obesity and Metabolic Syndrome

All three classes effectively reduce weight, with clear gradient in effect size: GLP-1 RAs (8–15%), dual agonists (15–20%), and triple agonists (22–25%)[1][2][4].

Hepatic steatosis improves across all classes, with dual and triple agonists showing greater liver fat reductions than GLP-1 RAs alone[3]. This is particularly relevant in South Asian populations exhibiting the "thin-fat" phenotype, where marked visceral adiposity and metabolic risk occur at relatively lower BMI thresholds[5]. Visceral adiposity and metabolic syndrome components (waist circumference, triglycerides, HDL-C, blood pressure, fasting glucose) also improve [1][2].

Type 2 Diabetes: Glycemic Control

GLP-1 RAs provide robust HbA1c reductions (1.0–1.8%), with low hypoglycemia risk [1]. Dual agonists offer superior glycemic control, consistently achieving HbA1c reductions of 2.0–2.5%, significantly outperforming GLP-1 comparators with greater weight loss and improved insulin sensitivity [1][2].

Triple agonists demonstrate HbA1c reductions of around 2.2% in phase 2 studies, showing modest incremental glycemic benefit over dual agonists but substantial advantages in weight reduction and metabolic risk markers[4].

Cardiovascular Outcomes:

GLP-1 RAs: Established Cardioprotective Benefit

Multiple large CVOTs in T2DM with high cardiovascular risk—including LEADER (liraglutide), SUSTAIN-6 (semaglutide), REWIND (dulaglutide), and HARMONY Outcomes (albiglutide) - show that GLP-1 RAs reduce major adverse cardiovascular events (MACE) including cardiovascular and all-cause mortality[3]. The landmark SELECT trial extended this benefit to patients with overweight/obesity and established CVD without diabetes, showing 20% MACE reduction (HR 0.80, 95% CI 0.72–0.90) at 40 months, confirming direct cardioprotective effects beyond glycemic control[3]. Current guidelines recommend treatment with GLP-1 RAs having proven CV benefit for patients with T2DM and established ASCVD or very high cardiovascular risk.

Dual GLP-1/GIP Agonists: Emerging but Promising Data

Tirzepatide demonstrates strong evidence for weight and glycemic efficacy across the SURPASS trial program (SURPASS-1 through SURPASS-5)[2]. The pivotal SURPASS-CVOT, completed in 2025, enrolled over 12,900 patients with T2DM and established ASCVD, showing noninferiority for 3-point MACE compared with dulaglutide (GLP-1 RA comparator) (HR 0.92, 95% CI 0.83–1.01), with significant all-cause mortality reduction (HR 0.84, 95% CI 0.75–0.94)[2][5]. Target trial emulation studies suggest 20% MACE reduction versus dulaglutide, driven primarily by all-cause mortality benefit[5].
Cardiovascular safety is established, and these drugs have potent effects on weight, visceral fat, blood pressure, and lipids. Dual agonists represent reasonable alternatives in CVD patients, particularly when weight and metabolic control are suboptimal on GLP-1 alone therapy, but these have not replaced GLP-1 RAs as default CV-protective incretin agents as of now.

Triple Agonists: Theoretical Promise Without Outcomes Evidence

Triple agonists possess compelling mechanistic rationale for cardiovascular benefit—maximal weight loss (24.2% in phase 2 retatrutide obesity trial), powerful effects on adiposity, blood pressure, lipids, insulin resistance, and liver fat[4]. However, completed large cardiovascular outcome trial data is still lacking. Phase 3 CVOTs for retatrutide are currently enrolling (expected completion 2027–2028). Their use in established CVD patients should currently be confined to clinical trial settings until robust cardiovascular and renal outcome data emerge[4].

Renal Outcomes and Cardiorenal Protection

GLP-1 RAs demonstrate proven reduction in albuminuria progression and slower eGFR decline in T2DM with CKD, thereby having strong recommendations in KDIGO/ADA guidelines as part of cardiorenal protective strategies, typically alongside SGLT2 inhibitors[1][5].

Dual agonists show promising renal signals through profound metabolic and hemodynamic improvements, but dedicated CKD outcome data are still emerging[2]. Triple agonists lack specific renal outcome evidence as of now; and renoprotective effects remain hypothetical pending trial completion[4].

For CVD patients with coexisting CKD, GLP-1 RAs currently possess the strongest evidence base for cardiorenal protection.

Safety and Tolerability

Gastrointestinal adverse effects (nausea, vomiting, diarrhea) are common across all incretin classes, particularly during initiation and dose escalation[1][2]. Dual and triple agonists may demonstrate slightly higher initial GI event rates versus GLP-1 RAs, though serious events appear comparable [2][4].

Intrinsic hypoglycemia risk remains very low across all classes especially when used without concomitant insulin or sulfonylureas[1][2]. Long-term safety data are extensive for GLP-1 RAs (>10 years), moderate for dual agonists (~5 years), and limited for triple agonists (phase 2–3 only)[1][2][4].

Table 1: Comparative summary of GLP-1, dual GLP-1/GIP, and triple agonists

Clinical Positioning: Phenotype-Driven Approach

Established ASCVD (with or without diabetes): GLP-1 RAs with proven CV benefit remain first-line incretin therapy, offering robust MACE and mortality reduction with established renal benefit.

T2DM, severe obesity, and high CV risk: When large, sustained weight loss and metabolic improvement are priorities—especially with marked visceral adiposity or MASLD—dual GLP-1/GIP agonists are a far better choice. GLP-1 RAs remain alternative options where dual agents are not feasible.

T2DM with CVD and CKD: GLP-1 RAs are preferred due to cardiorenal evidence and guideline endorsement. Dual agonists may eventually complement this space once dedicated outcome data mature.

Refractory severe obesity with multiple CV risk factors: Triple agonists show mechanistic promise and may become important in this niche but are currently regarded as investigational and used in research contexts.

Special Considerations for South Asian Populations

South Asian patients exhibit high cardiometabolic risk at relatively lower BMI thresholds due to central obesity, insulin resistance, and premature CVD[5]. GLP-1 RAs provide well-validated balance of CV protection and metabolic improvement. Dual agonists can be prioritized in severe obesity or MASLD phenotypes, taking cost considerations. Regardless of agent, cardiometabolic risk modification should be prioritized over weight loss alone. Fig 1 lists the various molecules in these classes of drugs and Table 1 gives a comparative summary of the three class of molecules.

Summary

For cardiovascular disease patients within the broader cardiovascular-metabolic spectrum, **GLP-1 RAs remain the cornerstone incretin therapy,** uniquely combining CVOT-proven MACE reduction, renal benefits, and extensive safety data. **Dual GLP-1/GIP agonists** offer superior weight and glycemic efficacy with promising CV and renal outcomes, making them ideal for carefully selected obesity-predominant and high-metabolic-risk phenotypes. **Triple agonists** currently represent a powerful but investigational frontier, with ongoing trials for long-term cardiovascular and renal safety and efficacy.

Therapy choice should be individualized, based on CVD severity, obesity grade, renal function, glycemic status, safety profiles, and cost considerations. Until dual and triple agonists demonstrate unequivocal superiority with more robust data, GLP-1 RAs continue to be used for cardiovascular protection in high-risk patients.

Fig 1: Classification of GLP1 based therapies

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Domain	GLP-1 RAs	Dual GLP-1/GIP	Triple GLP-1/GIP/Glucagon
Examples	Exenatide, Dulaglutide, Liraglutide, Semaglutide	Tirzepatide	Retatrutide
Receptors	GLP-1 only	GLP-1 + GIP	GLP-1 + GIP + Glucagon
HbA1c reduction	1.0–1.8%	2.0–2.5%	2.2% (phase 2)
Weight loss	8–15%	15–20%	22–25% (early trials)
Insulin resistance	Moderate improvement	Marked improvement	Marked improvement
Visceral fat	Yes	Greater than GLP-1	Maximal (early evidence)
NAFLD/MASLD	Proven reduction	Greater reduction	Strong early benefit
CVOT (MACE)	Proven reduction	Noninferior/possible superior	No CVOT yet
CV mortality	Yes (select agents)	Yes, Emerging evidence	Not yet studied
Renal outcomes	Proven albuminuria/eGFR benefit	Emerging benefit	No data
CKD usability	Safe to eGFR <15	Safe; data evolving	Unknown
Guidelines	Strong recommendation (ADA/KDIGO)	Increasingly preferred	Not yet recommended
Hypoglycemia	Very low	Very low	Very low
GI adverse effects	Common (nausea, vomiting)	Similar or higher initially	Similar; long-term pending
Long-term safety	Extensive (>10 years)	Moderate (5+ years)	Limited (phase 2–3)
Regulatory status	Approved worldwide	Approved worldwide	Investigational

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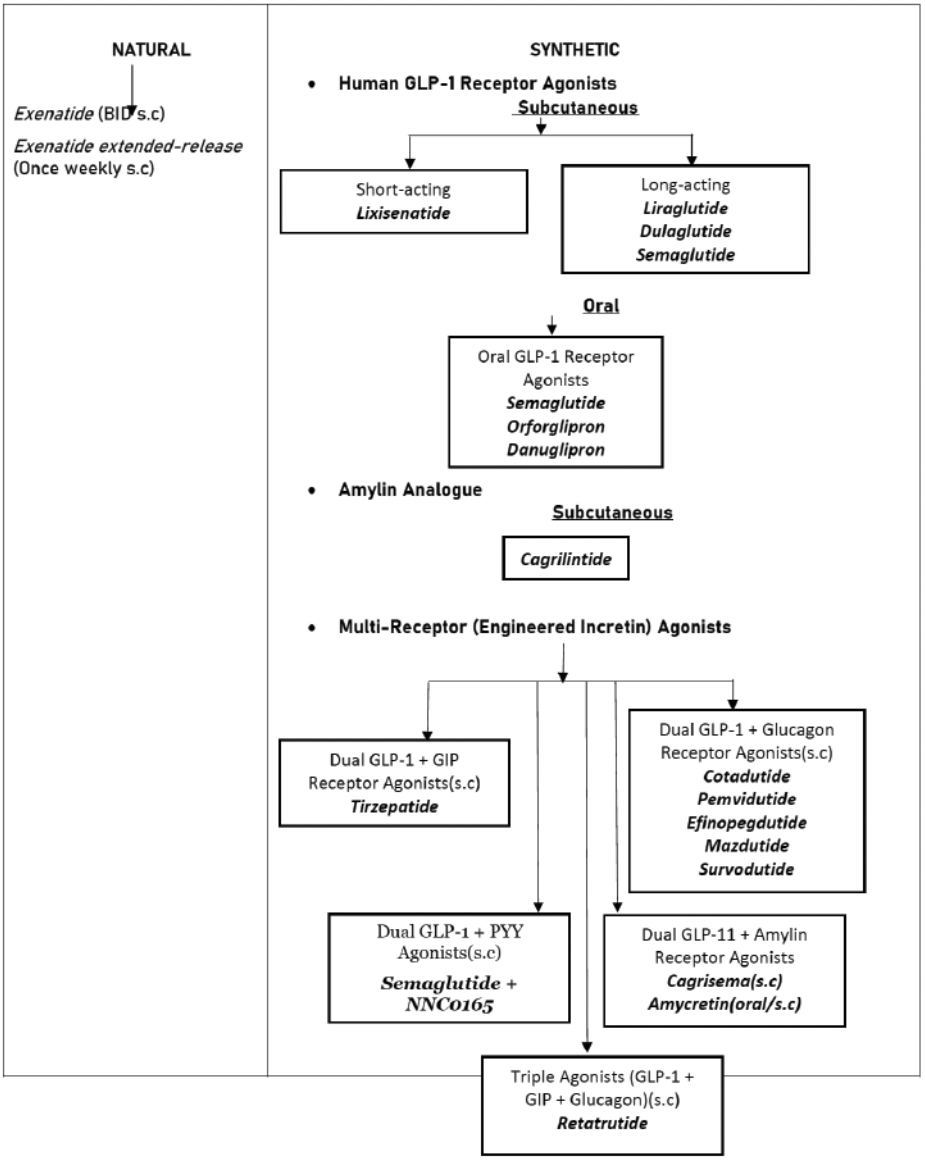

Combination Therapy in Pulmonary Arterial Hypertension: Earlier Is Better

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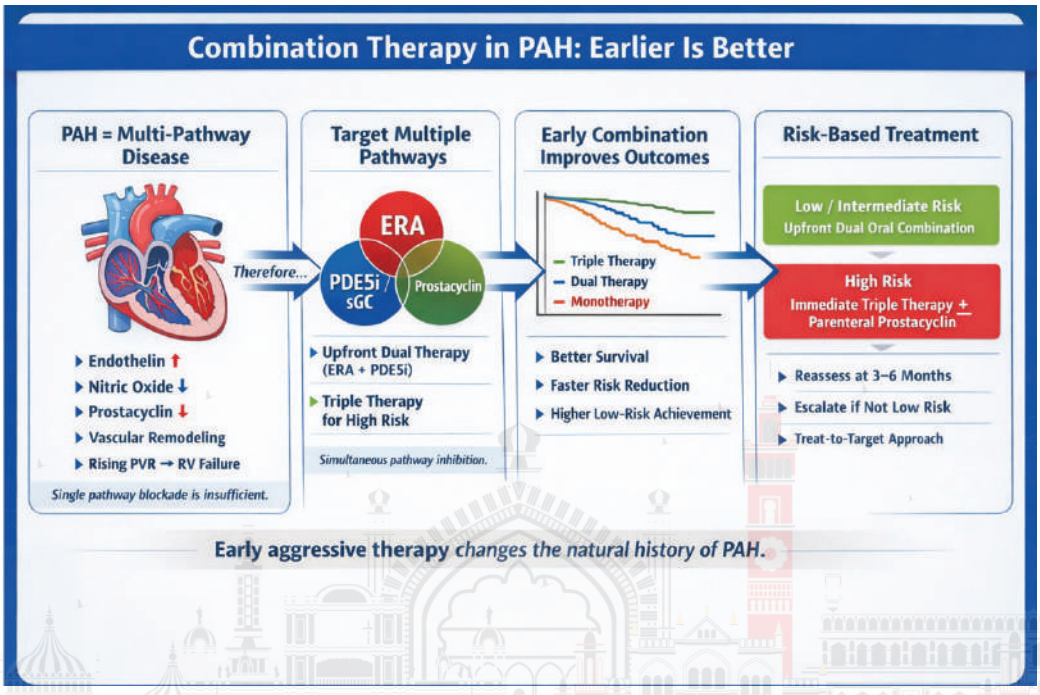
Pulmonary arterial hypertension (PAH) is a progressive and life-threatening disorder characterized by increased pulmonary vascular resistance, culminating in right ventricular failure and premature mortality. Despite therapeutic advances, PAH remains incurable, and long-term outcomes depend critically on early diagnosis and prompt initiation of effective therapy.1 Current pathobiological understanding identifies PAH as a multi-pathway disease driven by dysregulation of endothelin, nitric oxide, and prostacyclin signalling, leading to vasoconstriction, vascular remodelling, and inflammation. This complexity provides the biological rationale for combination therapy as the foundation of contemporary PAH management.[2]

paradigms favoured stepwise escalation, initiating monotherapy and intensifying only after clinical deterioration. This strategy permits ongoing pulmonary vascular remodelling and progressive right ventricular dysfunction during the early disease phase. Accumulating evidence now demonstrates that delayed treatment intensification adversely impacts prognosis, whereas early multi-target therapy modifies disease trajectory. Consequently, upfront combination therapy has replaced sequential escalation as the preferred approach.[3] Simultaneous inhibition of multiple pathogenic pathways underpins the therapeutic benefit of combination therapy. Monotherapy inadequately suppresses vascular proliferation and remodelling, allowing parallel mechanisms to persist. Dual and triple regimens achieve more rapid reductions in pulmonary vascular resistance, improve right ventricular–pulmonary arterial coupling, and increase the probability of attaining a low-risk clinical profile.[4] Early intervention is therefore essential to prevent irreversible pulmonary vascular and right ventricular structural changes.

Randomized trials and real-world registries consistently support this paradigm. Upfront dual oral therapy, typically combining an endothelin receptor antagonist with a phosphodiesterase-5 inhibitor or soluble guanylate cyclase stimulator, reduces clinical worsening and improves functional capacity compared with monotherapy. Importantly, registry data demonstrate that initial treatment strategy exerts a durable influence on long-term survival. Patients commenced on combination therapy exhibit superior outcomes relative to those treated with monotherapy, while triple therapy confers the greatest survival benefit, particularly among intermediate- and high-risk populations. These findings underscore that early therapeutic decisions shape prognosis over years rather than merely short-term clinical status.

Contemporary PAH management is anchored in structured risk stratification integrating clinical parameters, exercise capacity, biomarkers, imaging, and invasive hemodynamics. The principal therapeutic objective is rapid achievement and sustained maintenance of a low-risk profile, which correlates strongly with survival. Current risk-based algorithms recommend upfront dual oral combination therapy for patients presenting at low or intermediate

Graphical Abstract



risk. For high-risk patients, defined by severe symptoms, impaired exercise tolerance, or adverse hemodynamics, Immediate Triple Therapy, incorporating prostacyclin in addition to dual oral agents, is advised. Treatment response should be reassessed within three to six months, and failure to achieve low-risk status mandates prompt escalation.[5]

This treat-to-target framework emphasizes proactive optimization rather than reactive intensification. Delayed escalation allows continued vascular remodelling and progressive right ventricular impairment, diminishing the likelihood of meaningful recovery. In contrast, early aggressive therapy accelerates hemodynamic improvement, facilitates reverse remodelling, and favourably alters long-term survival trajectories.[5] Concerns regarding tolerability have historically limited early adoption of combination therapy. Although vasodilator agents may produce additive adverse effects such as headache, hypotension, and peripheral oedema, contemporary experience indicates that most patients tolerate dual oral therapy well, with manageable side-effect profiles through careful titration and monitoring. Early referral to specialized PAH centres is pivotal for accurate risk assessment, individualized therapeutic selection, patient education, and timely access to advanced treatments. The emergence of novel agents and improved prostacyclin delivery systems further expands therapeutic options, enhancing the feasibility of early multi-drug strategies across diverse clinical settings.[6]

In summary, PAH outcomes are determined early in the disease course. Pulmonary vascular remodelling and right ventricular adaptation precede overt clinical deterioration, rendering delayed intensification a missed opportunity to influence disease biology. Upfront combination therapy has become the standard of care, with Immediate Triple Therapy reserved for high-risk patients. Initial treatment strategy represents a major determinant of long-term outcomes, highlighting that timing itself constitutes a therapeutic intervention. In PAH, the critical question is no longer whether to combine therapies, but how early to initiate them. Adopting an "earlier is better" approach offers the greatest opportunity to improve survival and quality of life in patients with pulmonary arterial hypertension.

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Immunization in Heart Failure-An Update

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Heart failure (HF) is increasing in prevalence worldwide because of improved cardiac care as well as increasing longevity and associated with high morbidity and mortality. More than 50% of HF patients die within five years of diagnosis, so there is an unmet need to improve prognosis. HF is a chronic syndrome characterized by acute exacerbations. Respiratory infections are one of the most important factor responsible for exacerbation of underlying HF. In the OPTIMIZE-HF registry, respiratory infections accounted for about 15% of acute decompensations and were associated with a 60% higher in-hospital mortality.

Inflammatory processes are strongly activated during respiratory infections. Endothelial damage and coagulation dysregulation due to inflammation often precipitate for acute atherosclerotic events. Pneumonia and sepsis can cause myocardial injury and depress cardiac function leading to cardiac dysfunction and HF and this risk is more pronounced in patients with a history of HF.HF is associated with an increased risk of venous thrombo-embolism, and respiratory infections further elevate the risk of thrombotic related events. Most of respiratory acute infections can destabilize CVD and promote acute decompensation, and increase CV mortality.

The underlying pathogenic organisms of respiratory infections are predominantly influenza viruses and pneumococcal bacteria. Currently, a purely causative relationship between HF and influenza and/or pneumococcal infection has not been established.
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However, frequent, infection-induced HF exacerbations that follow a seasonal, periodic fashion imply an association between those organisms and the development and progression of HF.

Vaccination against respiratory infections in HF patients could serve as a potential cost-effective intervention to improve patients' quality of life and clinical outcomes Infective disorders may worsen HF symptoms and be a precipitant factor for AHF. The remarkable advances in the field of infectious diseases have gifted us with highly effective and safe vaccines to combat infections, especially respiratory infections. Influenza vaccination is associated with a reduced risk of all-cause death in patients with HF in observational studies and retrospective analyses. Influenza and pneumococcal vaccination, as well as COVID-19 vaccination, when available, should be considered in patients with HF.

The ESC guidelines recommend annual influenza vaccinations for patients with established CVD. The latest guidelines on HF and CAD have reinforced this recommendation as vaccination against influenza has proven safe. In the USA and UK, pneumococcal vaccination is recommended in patients older than 65 years (left for the health care professional's opinion to decide whether to vaccinate or not in the USA). It is also recommended earlier in high-risk immunocompetent patients, such as those with chronic cardiovascular disease (except hypertension). Based on expert opinion, the European Society of Cardiology recommends pneumococcal vaccination in patients with HF.

In conclusion, inflammation and infection are strongly associated with HF. Vaccination against influenza, pneumococcus, RSV, COVID-19, and other non-respiratory agents (including Shingles) are crucial goals for patients with HF, although largely underutilized. Vaccination could offer simple, affordable protection for this vulnerable population and may be game changer in reducing the morbidity and mortality of HF.

Aldosterone Synthase Inhibition: An Emerging Pathway in Hypertension Management (From receptor blocker to synthesis inhibition)

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Hypertension is a leading preventable cause of morbidity and mortality globally, affecting approximately 1.2 billion individuals worldwide, including around 200 million people in India, which accounts for up to 30% of the population. A variety of therapeutic options are available for managing hypertension, such as diuretics, beta-blockers, calcium channel blockers, angiotensin receptor blockers, angiotensin-converting enzyme inhibitors, alpha-blockers, centrally acting alpha receptor agonists and vasodilators. Despite the availability of these treatments, the prevalence of hypertension continues to rise, and a significant portion of the hypertensive population remains resistant to conventional therapies. Treatment-resistant hypertension is characterized by blood pressure that remains above therapeutic goals despite the use of three antihypertensive drugs, including a diuretic, at full doses, or the need for four or more antihypertensive medications. The prevalence of resistant hypertension is approximately 10–15% among hypertensive patients, increasing the risk of stroke, kidney disease, and cardiovascular disease.

Elevated aldosterone levels are observed in up to 10% of drug-naïve hypertensive patients. Additionally, aldosterone escape is common in patients receiving renin-angiotensin-aldosterone system (RAAS) blockers, such as ACE inhibitors, ARBs, and mineralocorticoid receptor antagonists (MRAs), leading to increased aldosterone levels. Aldosterone, synthesized in the adrenal zona glomerulosa by aldosterone synthase (CYP11B2), facilitates sodium reabsorption via epithelial sodium channels (ENaC) and induces volume expansion, vascular inflammation, and fibrosis, thereby increasing cardiovascular morbidity.

Drugs targeting aldosterone pathways include steroidal MRAs, such as spironolactone, which block aldosterone receptors and reduce mortality. However, these agents are associated with adverse effects, including hyper hyperkalemia (5-15%) and gynecomastia. Nonsteroidal options like finerenone mitigate some risks but do not suppress aldosterone production. Furthermore, patients on above drugs can also have increased levels of serum aldosterone levels because of escape.

Aldosterone synthase inhibitors (ASIs) are oral agents that selectively block aldosterone synthase (CYP11B2), effectively reducing aldosterone levels by 70-90%. This upstream intervention helps prevent the phenomenon known as "aldosterone escape." A potential concern with inhibiting aldosterone synthase (CYP11B2) in the treatment of hypertension is the possible suppression of adrenocorticotropic hormone (ACTH)-mediated cortisol secretion from the adrenal cortex, given that aldosterone synthase (CYP11B2) shares 93% identity with the enzyme responsible for cortisol production (CYP11B1). Therefore, highly selective agents are preferred to minimize side effects.

Common ASIs include:
- First Generation: Osilodrostat, which has limited selectivity and is primarily used for Cushing's syndrome.
- Second Generation: Baxdrostat and Lorundrostat, which exhibit over 300-fold selectivity for CYP11B2.

In a phase 1 clinical trial, Baxdrostat demonstrated safety and efficacy, reducing aldosterone levels by 51% to 73% at doses ranging from 0.5 to 5 mg/day in healthy normotensive individuals. The phase 2 BrigHTN trial involved patients with blood pressure greater than 130/80 mm Hg who were on stable doses of three antihypertensive medications. Participants received either a placebo or Baxdrostat at doses of 0.5, 1, or 2 mg/day for 12 weeks. The phase 3 BaxHTN trial revealed that among patients with uncontrolled or resistant hypertension, the addition of Baxdrostat to existing therapy resulted in a significantly lower systolic blood pressure (approximately 8-11 mm Hg) at 12 weeks compared to placebo, with a dose-dependent response

Comparison of MRAs and ASIs:

Feature	MRAs (e.g., Spironolactone)	ASIs (e.g., Baxdrostat)
Site of Action	Mineralocorticoid Receptor	CYP11B2 Enzyme
Aldosterone Levels	Increase (Reactive)	Decrease (Direct)
Hyperkalemia	High Risk	Moderate/Lower Risk
Hormonal SEs	Gynecomastia (Spironolactone)	None
Primary Limitation	Progesterone/Androgen cross-reactivity	Potential Cortisol suppression (if non-selective)

Safety and Tolerability
Potassium: Hyperkalemia is the most frequently reported adverse event, yet it is generally manageable. The incidence in Phase 3 trials was approximately 5–10% at therapeutic doses.

Cortisol: No clinically significant cortisol suppression has been observed with second-generation, highly selective agents at target doses.

Hyponatremia: Occurred in a small percentage of patients, necessitating monitoring in those receiving high-dose diuretics.

Conclusion
Aldosterone Synthase Inhibitors represent the first new class of antihypertensives in nearly two decades, offering a more precise, upstream approach to managing the renin-angiotensin-aldosterone system (RAAS). Clinical data support their use as a potent option for treatment-resistant and obesity-related hypertension, with potential applications in heart failure management.

Revascularisation in Ischemic LV Dysfunction - What have we Learned from RCTs?

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Ischemic left ventricular dysfunction is the most common cause of dilated cardiomyopathy of ischemic type and is associated with high morbidity and mortality. Revascularisation either by angioplasty or surgery (CABG) is a contentious issue and requires close examination of not only patients clinical variables, but also available scientific evidences from RCTs. Some of the older practices of routine revascularisation in ischemic cardiomyopathy is in conflict with the recent scientific data from randomised trials.

While STICH trial, which was a randomised study done to determine whether CABG reduced the risk of a primary and point of all-cause death in patients with ILVD receiving medical therapy, this study showed similar mortality in both surgical and medical arms.

Given the evidence of benefit in STICH across several secondary end points, an extension of STICH study was undertaken which at 9.8 years showed fewer patients in the CABG group died compared to medical treatment, specially in young patients.

Another study, revised – BCIS trial, which was done to study the benefit of PCI in patients of ILVD on medical therapy. This study showed primary event equally in both groups and there was no difference in key secondary end points of CV death or LVEF at 6 or 12 months.

Meticulous planning in eligible patients for revascularisation is needed in patients of ischemic cardiomyopathy, giving attention to shared discussion, including non-technical factors and considerations for achieving complete revascularisation with a special emphasis on mechanical circulatory support.

Recent ongoing trials will refine revascularisation strategy. For PCI (Restore – PCI, Improve – ICMP) or CABG (MASS-4HF) and will also compare above revascularisation modalities to contemporary optimal GDMT. Also CABG versus PCI head to head – STICH – 3 consortium will determine optimal revascularisation modality in ILVD with clear identifications CHIP-BCIS3 and PROTECT IV aim to define the role of elective PLVAD in complex PCI with ILVD.

Current Status of Bioresorbable Scaffolds in Coronary Interventions

Satish Suryavanshi

Drug-eluting stents (DES) have several limitations. Persistent issues include vascular inflammation, local thrombus formation, loss of vasomotor tone, and restenosis of the stented segment. Occasional late and very late stent thrombosis, stent fatigue fracture, unsuitability of stented segments for future surgical revascularization, impairment of vasomotor function, and jailing of side branches remain unresolved problems.

To address these limitations, **bioresorbable scaffolds (BRS)** were developed. **Bioabsorbable vascular scaffolds (BVS)** were first introduced in 2006. Similar to DES, neointimal proliferation is prevented by BVS through the release of antiproliferative drugs.

These novel devices have the potential to become suitable targets for future surgical revascularization, comparable to native vessels.

Absorb BVS and Other Scaffold Technologies

The prototype and most widely used scaffold was the **Absorb BVS,** consisting of a poly-Llactide (PLLA) frame coated with a 1:1 mixture of everolimus and an amorphous poly-D,Llactide matrix. The device is not radio-opaque; therefore, two radio-opaque markers are embedded at each end. It requires storage at 2–8°C.

The first version demonstrated significant recoil at 6 months and was subsequently replaced by version 1.1. This version had a strut thickness of 150 µm, an out-of-phase sinusoidal hoop design with multiple links, and a crossing profile of 1.4 mm. The absorption time was less than 3 years.

Other BRS platforms have used materials such as poly-salicylic acid, tyrosine polycarbonate, magnesium, or other metals. These materials were used in devices such as **DESolve, Igaki- Tamai, and Amaranth.** The **Reva ReZolve** scaffold used deca-amino-tyrosine polycarbonate combined with sirolimus. Biotronik incorporated magnesium instead of PLLA in its absorbable metal stents (AMS) and DREAMS platforms.

Evidence from ABSORB Trials

Results from the ABSORB trials demonstrated that BVS was comparable to the available DES of that time. **BVS version 1.1** was used in Cohort B patients and showed good late clinical outcomes, with no scaffold thrombosis at 3 years and a major adverse cardiac event (MACE) rate of 10.1%. These findings were supported by the EXTEND study.

Notably, MACE rates remained stable after scaffold resorption (2–3 years), even up to 5 years. **ABSORB II** compared BVS with an equivalent metallic stent in 501 patients. **ABSORB III,** published in 2015, enrolled 2,008 patients across multiple centers and led to FDA approval in the USA. However, device thrombosis was approximately double in the BVS group compared to the metallic stent group. Subsequent analyses also revealed higher rates of subacute scaffold thrombosis.

ABSORB IV, an extension of ABSORB III, was presented at TCT 2018. It showed that 30-day and 1-year rates of target vessel failure and angina were non-inferior to DES.

Registry Data and Later Observations

The **GHOST-EU** registry reported a definite scaffold thrombosis rate of 2.1% at both 6 and 12 months. Other BVS registries that included more complex lesions also demonstrated a higher 1- year stent thrombosis rate compared to second-generation DES.

Concurrent studies of polymer-based BRS, including **DESolve, ARTS18AZ, Reva, and REZOLVE,** were conducted. **PROGRESS-AMS** was the first prospective trial evaluating metallic (magnesium-based) BVS. Although acute results were comparable to other stents, reduced radial strength resulted in high restenosis rates.

Challenges and Concerns

The initial enthusiasm for BVS technology declined sharply following reports of subacute and late scaffold thrombosis. Reevaluation highlighted the need for meticulous case selection and modified implantation techniques. Additional concerns include:

1. **Thicker and wider struts,** which may protrude into the lumen, disturb laminar flow, and promote platelet activation.
2. **Higher incidence of acute scaffold thrombosis,** necessitating validation of optimal antiplatelet strategies.
3. **Delayed re-endothelialization** and healing influenced by antiproliferative drugs.
4. **Lack of clear superiority** over current-generation DES; short-term outcomes are comparable between the two.

New-Generation Bioresorbable Scaffolds

Due to high scaffold thrombosis rates, **ABSORB GT1** was withdrawn from the market. Newer designs with improved materials were subsequently developed. Limitations of first-generation scaffolds included insufficient ductility, low tensile strength, and thrombogenicity related to lactic acid release during degradation—contributing to late and very late thrombosis.

New-generation devices aim to overcome these issues. Scaffolds such as **Magmaris, Xinsorb, NeoVas, Firesorb, MeRes 100, Mirage, Bioheart, Fantom Scaffold, Credence BtK (Meril LS), and Motiv** have been introduced. These devices feature thinner struts (~100 µm), hybrid geometry with a PLLA backbone, and improved radial strength.

They have a crossing profile of approximately 1.2 mm and include radio-opaque markers at both ends to enhance visibility. Radial strength is approximately 1.2 bar with low recoil (~1.6%) and minimal balloon overhang. These scaffolds allow greater over-expansion after deployment and have optimized drug degradation profiles. Unlike earlier devices, they can be stored at room temperature and are currently under active evaluation.

MeRes 100 delivers sirolimus at a dose of 1.25 µg/mm^2.
Implantation Technique (PSP Strategy)

Optimal BRS implantation requires:

- Adequate pre-dilatation
- Accurate vessel and device sizing (balloon inflation at 16–20 atm without waist)
- Proper post-dilatation (PSP technique: Pre-dilatation, Sizing, Post-dilatation)

To facilitate optimal PSP, the MeRes 100 scaffold is supplied with two non-compliant balloons:
one matching scaffold size and another 0.25 mm larger.
BRS implantation should generally be avoided in vessels <2.5 mm or >3.75 mm, long lesions, and heavily calcified lesions. Preferred use includes young patients with two-vessel disease, deployment of two BRS in a single vessel without overlap, and selected primary PCI cases.

MeRes-1 Trial
In the MeRes-1 trial, Seth et al. reported 3-year clinical outcomes in 108 patients treated with MeRes 100. The cumulative MACE rate was 1.87%, attributable solely to target lesion
revascularization (TLR). There were no cardiac deaths, myocardial infarctions, or scaffold thrombosis.

In Hybrid stenting BRS is used in proximal lesions and DES or drug-eluting balloons (DEB) are used in distal diffuse disease. This approach reduces overall metallic burden compared to fulllength DES implantation in long lesions.

Conclusion
BVS represents a novel and promising concept in coronary intervention, particularly with newergeneration devices. When long-term vessel patency and restoration of vasomotor function are desired, BVS offers a compelling option. Early setbacks have been largely addressed through improved device design and refined implantation techniques. BVS is expected to re-emerge as an important strategy in interventional cardiology. specially in young patients with moderate size coronaries.

Topic : AI in Cardiology : A Doctors Friend or Enemy

FEB 2026

Anurag Dhingra

Artificial intelligence has reached a decisive moment in clinical medicine. The question confronting cardiologists today is no longer whether AI will enter routine practice, but whether clinicians will lead its integration or be forced to react to it. At UP CSI 2026, the discussion on "AI for Doctors – A Doctor's Best Friend or Worst Enemy" reframed the debate in academic and clinical terms, moving beyond hype to examine where AI genuinely fits into cardiovascular care and daily clinical workflows

Why This Conversation Matters Now

Cardiology sits at the intersection of data intensity and time scarcity. Imaging, biomarkers, guidelines, comorbidities, and longitudinal follow-up generate volumes of information that exceed what any individual clinician can process in real time. At the same time, physicians face increasing administrative load, documentation requirements, and patient expectations. Data shared during the session highlighted that nearly 80% of doctors report feeling persistently overwhelmed, with minimal time for reflection, learning, or family life.

Against this background, AI is emerging not as a futuristic luxury, but as a response to structural inefficiencies in modern healthcare delivery. The urgency is amplified by patient behavior. Approximately 65% of patients now search online for medical information before consulting a doctor, and over half disengage during long-term treatment due to inadequate follow-up or lack of personalized communication. These realities demand systems that extend clinical care beyond episodic consultations.

Understanding AI Through a Clinical Lens

A key strength of the UP CSI session was its effort to demystify artificial intelligence for clinicians. Generative AI was positioned not as autonomous intelligence, but as a probabilistic system capable of producing text, images, and summaries based on learned patterns from vast medical and non-medical corpora. Large Language Models, trained on billions of documents, can interpret clinical questions, summarize guidelines, and assist in structured clinical reasoning when appropriately constrained.

The analogy offered resonated strongly with the audience: AI functions best as a highly efficient junior assistant—fast, consistent, and tireless—yet entirely dependent on the clinician for judgment, accountability, and ethical decision-making. This framing is essential to prevent both unrealistic expectations and unwarranted resistance.

AI in Action: Relevance to Cardiology Practice

Rather than focusing on abstract capabilities, the discussion emphasized practical use cases relevant to cardiology:

- Rapid summarization of evolving guidelines in lipid management, heart failure, and preventive cardiology
- Generation of patient education material tailored to Indian socio-cultural contexts and literacy levels
- Assistance in documentation, discharge summaries, and follow-up planning
- Clinical decision support that augments differential diagnosis without replacing physician reasoning

Findings from MediSage's national expert panel suggested that AI-enabled systems can potentially free up close to 90 minutes per clinician per day. Importantly, these gains arise not from reducing clinical rigor, but from eliminating repetitive cognitive and administrative tasks that add little clinical value.

Agentic AI and Longitudinal Care

One of the more forward-looking concepts introduced was agentic AI—systems designed to act proactively within predefined boundaries. In cardiology, this could translate into AI agents that support chronic disease management by tracking adherence, monitoring symptoms, prompting timely investigations, or preparing structured clinical summaries ahead of follow-up visits.

Such systems hold particular promise in conditions like hypertension, dyslipidemia, heart failure, and post-intervention follow-up, where continuity of care determines outcomes. However, the session emphasized that these agents must function under clinician oversight, with transparency and clear accountability, to maintain patient trust and clinical safety.

For cardiologists, this distinction is critical. AI can enhance pattern recognition and guideline adherence, but nuanced decisions—balancing comorbidities, patient preferences, and real-world constraints—remain inherently human responsibilities.

The Road Ahead

AI literacy will increasingly become part of professional competence, much like ECG interpretation or echocardiography.

AI is neither a panacea nor a peril by default. Its impact on cardiology will depend on how thoughtfully it is integrated into clinical workflows. The future will not belong to AI replacing cardiologists, but to cardiologists who understand AI well enough to use it safely, critically, and effectively.

UP CSI 2026 made one thing clear: artificial intelligence is best viewed not as an adversary to clinical judgment, but as a catalyst that can restore time, focus, and depth to the practice of medicine—if doctors remain firmly in the driver's seat.


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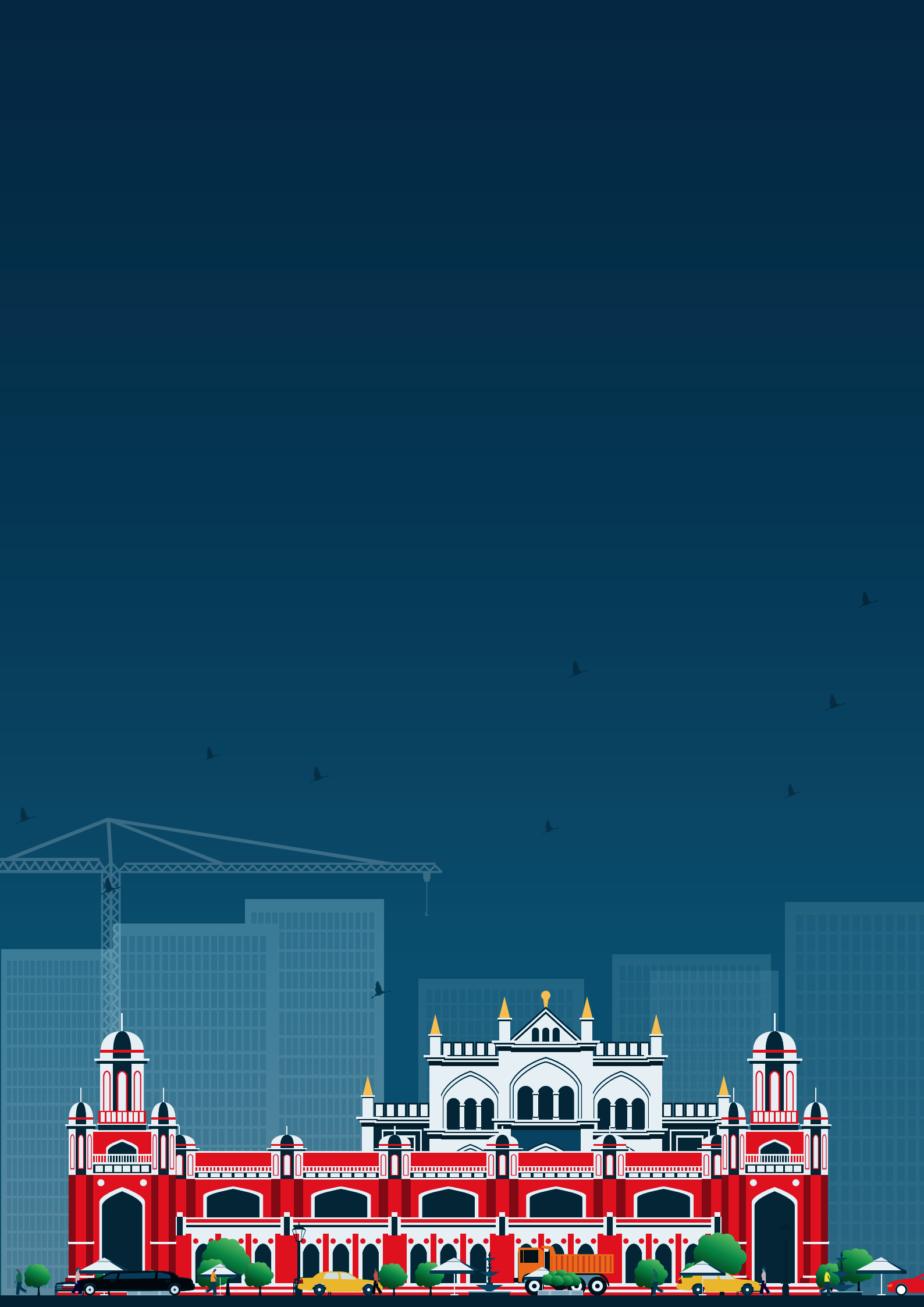


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