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EDITORIAL

First quinquennium completed: highlights from *EHJ Open* 2025

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In this editorial, the Editors present some of the scientific highlights from the past year that, in different ways, illustrate the breadth, diversity, and ongoing momentum of *EHJ Open*. These examples are not intended as an exhaustive catalogue of all the exceptional work published over the year but rather serve as a representative reflection of the journal's scientific scope and its place in today's cardiology landscape. Since many equally important contributions are not included here, we invite you to fully explore the journal's content, which is all open access and available to all.

Structural heart disease and valve innovation

Recent advances in structural heart disease that were published in *EHJ Open* last year highlighted a deepened integration of imaging, biology, genetics, and clinical decision-making.

Valvular heart disease is a fast-evolving branch of interventional cardiology, driven by innovation and cumulating clinical evidence. The timing of intervention in asymptomatic severe aortic stenosis is one of the topics for which benefits in truly low-risk individuals remain nuanced and require individualized interpretation.[1] After the EARLY TAVR trial showing that earlier intervention in asymptomatic severe aortic stenosis reduced major cardiovascular events by 50%,[2] current ESC/EACTS Guidelines state that intervention should be considered in asymptomatic severe, high-gradient AS with preserved LVEF as an alternative to close active surveillance when procedural risk is low.[3]

Findings from the Atherosclerosis Risk in Communities (ARIC) study[4] reported on associations of ankle brachial index (ABI), a marker of subclinical atherosclerosis, with coronary artery and extra-coronary vascular and valvular calcification in 1420 older participants.[5] A low ABI (≤0.9) had the strongest association with coronary calcium calcification and weakest with cardiac valve calcification, whereas a high ABI (≥1.3) tended to be more strongly associated with valvular calcification than with vascular calcification.[5] This suggests distinct pathophysiology of calcification across vascular beds and cardiac valves.[6] For the future perspectives in this area, medical treatments for aortic stenosis to control disease progression are now moving forward into true translation of experimental and observational studies into clinical trials.[7] The vitamin K-dependent matrix Gla-protein prevents calcium phosphate precipitation and crystallization and anticoagulant vitamin K antagonists have been associated with valvular calcifications, thereby raising the notion of vitamin K to prevent valvular calcification.[8] The randomized clinical trial PASSPORT evaluates vitamin K1 supplementation as a potential disease-modifying therapy for non-severe calcific aortic stenosis using CT-based quantification of valve calcium as the primary endpoint.[9] This opens up for novel avenues towards a long-awaited effective medical therapy to complement the present interventional solutions.

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A complete understanding of pathophysiological process and the identification of future therapeutic targets need to be taken into consideration because of the spatial heterogeneity of valvular disease-driving procceses.[10] To this end, combining CUBIC and SWITCH histological techniques[11] allows to render of calcified tissues transparent.[12] Application of the latter protocol to human calcified aortic valves revealed organized and heterogeneous valvular neuronal networks,[12] which represents a previously inaccessible anatomical layer for morphological analyses that expands the valvular pathobiology to neuro-immune biology.

Beyond the aortic valve, structural interventions for mitral regurgitation were clarified in a comprehensive meta-analysis comparing surgical mitral valve repair with mitral transcatheter edge-to-edge repair (m-TEER). Although mortality was comparable, surgery was associated with lower rates of heart failure rehospitalization and significantly fewer mitral valve reinterventions.[13] Finally, LTBP2 mutations were shown in both humans and mice to drive myxomatous mitral valve degeneration via dysregulated TGF-β signalling,[14] providing a clear molecular anchor for future perspectives in mitral regurgitation in addition to current precision strategies, advanced imaging, and novel transcatheter technologies.

Cardio-oncology complexities of bleeding risk in antithrombotic treatments

The Special Cardio-oncology Article Collection in the fifth volume of *EHJ Open* covered a breadth of how malignancies and their treatments may reshape both acute and chronic cardiovascular trajectories, as previously highlighted.[15] One of todays' major cardio-oncological challenges is the bleeding risk, which may represent a key clinical determinant guiding antithrombotic decisions.[16] Among patients admitted for STEMI receiving percutaneous coronary intervention in the UK between 2005 and 2019, cancer conferred a 50% increased risk of major bleeding compared with non-cancer patients after adjustment for baseline differences, pointing to mitigating bleeding risk in STEMI with cancer is of paramount importance to improve outcomes.[17] Complementing these findings, adding cancer as a binary variable to the five-point PRECISE-DAPT score[18] was recently shown to improve its discrimination ability.[19] In the first external validation cohort however, although PRECISE-DAPT Cancer Score categorized more cancer patients at high bleeding risk, this resulted in only marginal improvement for the discrimination of bleeding events compared with the original PRECISE-DAPT score without taking cancer into consideration.[20]

Inflammation and its resolution

In atherosclerotic cardiovascular disease (ASCVD), a residual inflammatory risk may exceed cholesterol-related risk in patients treated with statins.[21] Consistent with this, a real-world analysis of 39 638 adults with ASCVD who underwent routine hsCRP and LDL-C testing showed that elevated inflammatory risk, either alone or in combination with high LDL-C, was a stronger predictor of adverse outcomes than elevated cholesterol alone.[22] Advances in inflammation biology have extended the role of specialized pro-resolving mediators beyond the resolution of ASCVD-associated inflammatory processes,[23] to showing a similar orchestrating role in cardiac healing.[24] Dysregulation of these lipid-derived pathways may lead to a pathophysiological shift from adaptive cardiac repair to progression into heart failure.[24] These findings position inflammation-resolution as a therapeutic frontier extending beyond traditional anti-inflammatory strategies.

Heart failure, cardiomyopathies, and arrhythmias

Heart failure and cardiomyopathy research published in the fifth volume of *EHJ Open* highlighted the increasingly complex interplay between inflammation, electrophysiology, and metabolic derangements. Electrophysiologic remodelling also emerged as a crucial determinant of heart failure trajectories. In patients with heart failure, atrial fibrillation was associated with impaired VO_2 peak, potentially due to reduced stroke volume and/or arterio-venous oxygen difference, reinforcing atrial fibrillation as not only a comorbidity but also a direct amplifier of heart failure severity and a potential target for earlier rhythm-control strategies in selected patients.[25] In adult congenital heart disease (ACHD), secondary hyperparathyroidism[26] emerged as a surprisingly common comorbidity, observed in nearly 15% across the spectrum of ACHD and most common in complex and more severe disease, particularly those with predominant right heart involvement in the CHD-HYPER-study.[27] The 2025 ESC Guidelines for the management of cardiovascular disease and pregnancy endorsed the modified WHO (mWHO) classification for risk stratification,[28] a recommendation which received support from contemporary national registry data demonstrating an association between higher mWHO class and adverse maternal and obstetric outcomes in women with ACHD.[29]

In summary, the articles highlighted in this editorial underline the importance of gathering all cardiology subspecialities in one common forum to not only connect basic science and clinical research but also illustrate common pathways, for example, connecting heart failure with arrhythmias, the inflammation paradigm in atherosclerosis, and valvular heart disease at the intersection with imaging, interventional cardiology, and genetics. Having completed its first quinquennium, *EHJ Open* now confidently steps towards the horizon of a decade.

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There are no new data associated with this article.

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