BERZELIUS SYMPOSIUM 111

Acute and Chronic Heart Failure

11-13 March 2026 in Stockholm

Berzelius Symposium 111 is organized by the Swedish Society of Medicine in cooperation with the Swedish Society for Cardiothoracic Surgery.

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BERZELIUS SYMPOSIUM 111

Acute and Chronic Heart Failure

The Swedish Society of Medicine, together with the Swedish Society for Cardiothoracic Surgery, is pleased to announce Berzelius Symposium 111 – a multidisciplinary meeting focusing on acute and chronic heart failure. This symposium will bring together leading national and international experts to review and discuss the latest developments in diagnosis, treatment, and management of heart failure.

Target audience:
Cardiologists, internal medicine specialists, general practitioners, cardiothoracic surgeons, anesthesiologists, intensive care clinicians, and other professions interested in the field.

Key themes:
- Heart failure remains associated with very high morbidity and mortality
- Management and treatment are multidisciplinary and require close coordination
- Optimal care involves a broad spectrum of diagnostic and therapeutic approaches
- Collaboration across specialties is essential for improved outcomes

The program will run from Wednesday to Friday, featuring case-based discussions, expert presentations (15–20 minutes with discussion), and a symposium dinner on Wednesday evening.

We warmly welcome you to join us for this important and inspiring symposium!

Program Day 1

Moderators: Göran Dellgren and Styrbjörn Blohmé

07.30-08.00 **Coffee**

08.00-08.15 **Welcome, Introduction of day**
Case presentation by Styrbjörn Blohmé, Stockholm

08.15-08.45 **Definitions, types (HFrEF, HFpEF), etiologies leading to Heart Failure HF**
Maria Crespo-Leira, Cardiology, A Coruña, Spain

08.45-09.15 **Epidemiology, molecular mechanisms and genetics in chronic heart failure?**
Gustav Smith, Professor and senior consultant in cardiology, Gothenburg University and Sahlgrenska University Hospital, Gothenburg, Sweden

09.15-09.45 **Congestive Heart Failure, what are the diagnostic challenges?**
Gerhard Wikström, Cardiology, Uppsala

09.45-10.15 **Break and coffee**

10.15-10.45 **Arrhythmias and congestive heart failure**
Frieder Braunschweig, Cardiology, Karolinska, Stockholm

10.45-11.15 **Why Some Hearts Adapt and Others Fail - Exploring the variability of the failing heart**
Kristjan Karason, MD, Transplantation Center, Sahlgrenska University Hospital, Gothenburg

11.15-11.30 **Case Unveiling Presentation**

11.30-12.30 **Lunch**

Diagnostic Section in CHF

Case presentation by Emely Ögren, Gothenburg

12.30-13.00 **Imaging with echocardiography in CHF**
Maria Eriksson, Associate professor, Clinical Physiology, Dpt Molecular Medicine and Surgery, Karolinska Institutet, Stockholm

13.00-13.30 **Why are biomarkers important in CHF – the evidence and how I use them?**
Dr Kieran Docherty, Senior Clinical Lecturer and Honorary Consultant Cardiologist, School of Cardiovascular and Metabolic Health, University of Glasgow, Scotland

13.30-14.00 **Investigation of specific cardiomyopathies and therapeutic implications**
Arash Mokhtari, Associate Professor, Consultant Cardiologist, Heart Failure Section, Skåne University Hospital

14.00-14.30 **A pathologist's perspective on pathophysiology in Congestive Heart Failure**
Anders Oldfors, Professor/Senior Consultant, Clinical Pathology, Sahlgrenska University Hospital, Gothenburg

14.30-15.00 **The Value of MR in Congestive Heart Failure**
Marcus Carlsson, Physiology, Karolinska, Stockholm

15.00-15.30 **Break and coffee**

15.30-16.00 **Right heart failure and interplay with left heart failure – myth or reality?**
Marco Astengo, Sahlgrenska, Göteborg

16.00-16.30 **Tricuspid insufficiency and right ventricular strain in heart failure?**
Per Lindqvist, Cardiology, Umeå

Treatment of Chronic Heart Failure

16.30-17.00 **What have we learned from the Swedish Heart Failure Registry?**
Charlotta Ljungman, Cardiology, Sahlgrenska, Göteborg

17.00-17.30 **Heart Failure and valve diseases – when is intervention or surgery preferred?**
Anders Jeppsson, Surgery, Sahlgrenska, Göteborg

17.30-17.50 **CHF and devices – which patient, when and how?**
Cecilia Linde, Cardiology, Karolinska, Stockholm

17.50-18.00 **Case Unveiling Presentation**

18.00 **Photography**

18.15 **Drink, Mingel and Symposia Dinner at the Swedish Society of Medicine**

Program Day 2

Moderators: Lena Jideus and Nadia Sandström

07.30-08.00 **Coffee**

08.00-08.15 **Introduction of day**
Case presentation by Nadia Sandström, Örebro

Treatment of Chronic Heart Failure, cont

08.15-08.45 **Clinical Assessment of Advanced Heart Failure – When to Consider Advanced Therapies**
Kristjan Karason, MD, Transplantation Center, Sahlgrenska University Hospital, Gothenburg

08.45-09.15 **CHF and Drugs – what Evidence and for what?**
Lars Lund, Cardiology, Karolinska, Stockholm

09.15-09.45 **PCI or CABG in CHF and CAD – when to choose what?**
Elmir Omerovic, Cardiology, Sahlgrenska, Göteborg

09.45-10.15 **Break and coffee**

10.15-10.45 **Percutaneous mitral and tricuspid devices in heart failure – when and in whom?**
Torsten Doenst, Surgery, Jena, Germany

10.45-11.15 **Heart Transplantation and LVAD Bridge-to-HTx – why is it considered gold standard?**
Göran Dellgren, Surgery, Sahlgrenska, Gothenburg

11.15-11.45 **The recent evolution or revolution of heart transplantation – how did we become the largest institution in the world and how will the future look like?**
Ashish Shah, Surgeon, Vanderbilt University, Nashville, USA

11.45-12.15 **Myocardial Recovery: Future Challenges and Opportunities for MCS**
Mandeep Mehra, Cardiology, Harvard University, Boston, USA

12.15-12.20 **Case Unveiling Presentation and HTx patient perspective**

12.20-13.20 **Lunch**

How to optimize care in hospital and in primary care for CHF patients

13.20-13.20 *Case presentation by Jonathan Johansson, Göteborg*

13.20-13.30 **Bridging the gap between the GP and the cardiologist - better Heart Failure Care Starts in Primary Care**
Veronica Milos Nymberg, General Medicine, Malmö

13.50-14.10 **Collaboration between hospital heart failure clinics and primary care: key factors to success?**
Jonas Spaak, Cardiology, Stockholm

14.10-14.30 **CHF patients is best managed by nurses – this is how we do it**
Maria Liljeroos, RN, Associate Professor, Mälarsjukhuset Eskilstuna and Linköping university.

14.30-14.45 **Panel discussion with speakers and moderators**

14.45-15.15 **Break and coffee**

15.15-15.35 **What is the evidence for fluid and salt restrictions in CHF patients?**
Linn Höög, RN, MSc, PhD-student, Department of Cardiology, Heart and Vascular Center, Karolinska University Hospital, Sweden, Department of Neurobiology, Care Sciences and Society, Karolinska Institute Sweden

15.35-15.55 **Telemonitoring in Advanced Heart Failure - Clinical benefits, personal experience, and ongoing research**
Thomas Rydenstam Mellberg, MD PhD, Heart Failure Specialist, Department of Thoracic Surgery and Cardiology, Sahlgrenska University Hospital

15.55-16.15 **This is how I work up severe heart failure patients – and where we find them?**
Oscar Braun, Cardiology, Skåne University Hospital, Lund

16.15-16.45 **Panel discussion with speakers and moderators and case unveiling presentation**

Ongoing Scandinavian Studies Impacting CHF Patients

16.45-17.45 **Rapid fire presentation - on-going studies - 10 min presentation, 5 min discussion**

STICH Swedeheart study - Björn Redfors, Cardiology, Sahlgrenska, Göteborg
Comparison PCI vs CABG with HFrEF and CAD

SweVAD study - Göran Dellgren, Surgery, Sahlgrenska, Göteborg
RCT between medical management and LVAD in CHF

SPIRIT-HEFpEF study- Lars Lund, Cardiology, Karolinska, Stockholm
Spironolactone Initiation Registry Randomized Interventional Trial in Heart Failure with Preserved Ejection Fraction

SCAPIS study – Göran Bergström, Physiology, Sahlgrenska, Göteborg
How can the SCAPIS study increase understanding of Heart Failure?

17.55 **Resolution of the day**

18.00 **Mingel**

19.00-22.00 **Faculty Dinner**

Program Day 3

Moderators: Sigurdur Ragnarsson and Emilia Johansson

07.30-08.00 **Coffee**

08.00-08.15 **Introduction**
Case presentation by Emilia Johansson, Lund

Acute Heart Failure and Cardiogenic Shock

08.15-08.45 **Beyond the Pump: Reframing Cardiogenic Shock in Heart Failure?**
Mandeep Mehra, Cardiology, Harvard University, Boston

08.45-09.15 **Acute heart failure management in the intensive care unit – how and for whom?**
Mattias Törnudd, Anesthesia, Linköping

09.15-09.45 **Cardiogenic shock in the ICU – how and when should we use ECMO?**
Michael Broomé MD PhD, Associate Professor, ECMO Centre, Karolinska University Hospital

09.45-10.30 Break and coffee

10.30-11.00 **Cardiogenic Shock Registry Outcomes - What's New and How to Improve**
Maryjane Farr, MD, MSc, Professor of Medicine, University of Pennsylvania, Section Chief, Heart Failure, LVAD, Heart Transplantation, Director of Heart Transplant Research, Penn Transplant Institute, Philadelphia, PA, United States

11.00-11.30 **ECPR a resource intensive treatment - for whom and when?**
Bengt Redfors, Anaesthesia, Sahlgrenska, Gothenburg

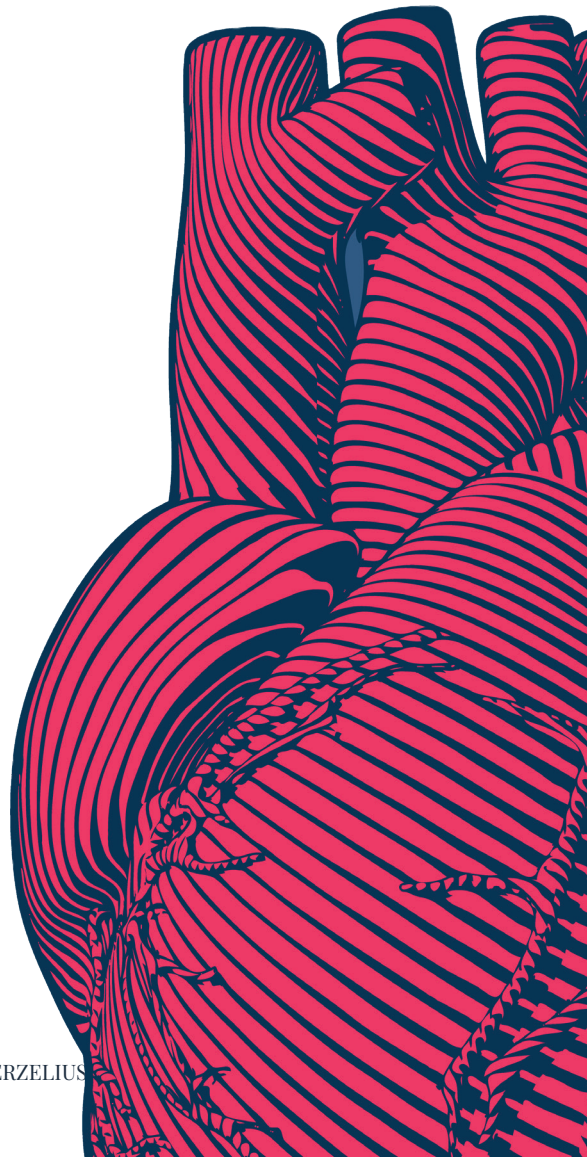
11.30-12.00 **Quality-of-life after ECMO and intensive care for acute heart failure – what's known?**
Inga lára Ingvarsdóttir, Anaesthesia, Reykjavik, Iceland

12.00-12.30 ***Acute heart failure and short-term MCS – what is the level of evidence for what***
Emil Najjar, Cardiology, Karolinska, Stockholm

12.30-12.45 **Case Unveiling Presentation and Resolution of Day, Conclude and Fairwell**

12.45-13.30 **Lunch**

Speakers abstracts page



ABSTRACTS

*Epidemiology, molecular mechanisms and genetics in chronic heart failure?**

Gustav Smith

Smith obtained a PhD in cardiovascular and genetic epidemiology from Lund University and undertook post-doctoral training focused on genetic and post-genomic studies at Harvard Medical School, Massachusetts General Hospital and the Broad Institute of MIT and Harvard.

In his research, he seeks to understand contemporary epidemiological trends in heart disease, underlying mechanisms on the molecular level with particular emphasis on myocardial disease and heart failure, and to contribute towards improved disease prediction, prognostication, prevention, therapy and ultimately outcomes in this patient group.

Towards these aims, the research group employs a diverse range of tools ranging from nation-wide registers, population-based and clinical cohorts, systematic molecular profiling platforms ('omics'), and clinical trials. He will provide updated epidemiological and pathophysiological perspectives on heart failure.

Congestive Heart Failure-which are the differential diagnoses?

Gerhard Vikström

Congestion has a central role both for presentation and prognosis for the patient with acute heart failure. One must remember that subclinical congestion is a driver of the symptoms and compensatory mechanisms such as RAAS activation. It is therefore important with high quality diagnostic work up and treatment according to findings and recommended guideline treatment.

The lack of a clear definition and classification of heart failure in the individual patient impairs both screening, detection and management of heart failure. Further, it is important to understand that congestion is not equal to volume overload.

There are several attempts to classify and simplify the definition of heart failure but in practice most of the attempts for classification and definition waive the ability for increasing the quality of follow up and diagnosis of a devastating disease. The present very popular view with diastolic and systolic heart failure defined from Ultrasound examinations with studies of both volumes and tissue of the heart has improved the understanding of the heart failure syndrome. After these observations were made some new medications has improved the possibility for medical treatment for a number of patients.

John Cleland suggested in 2022 a criterium for rule in and rule out heart failure with the help of natriuretic peptides. In addition a pathologic ECG should enforce the probability of heart failure.

NT-proBNP and prognosis
NT-proBNP levels are important prognostic factors for both DNHF and ADHI (acute HF). Notably, patients with ADHI had consistently higher risks than those with DNHF with the same NT-proBNP level for 1-year all-cause mortality.
int j cardiol2022 Sep 15:363:163-170

Arrhythmias and congestive heart failure

Frieder Braunschweig

Heart failure compromises not only the contractile function of the heart but also its electrical system. Heart failure and arrhythmias have a bidirectional relationship in which each condition can induce or aggravate the other. Patients with heart failure typically exhibit dilated cardiac chambers, myocardial scarring and fibrosis, as well as neurohormonal and sympathetic activation, all of which provide substrates and modulators for arrhythmias.

Accordingly, atrial fibrillation, the most common cardiac arrhythmia with a steadily increasing incidence, is highly prevalent among patients with heart failure ranging from 30 to 60%. In these patients, atrial fibrillation further deteriorates hemodynamics, increases the symptomatic burden and contributes to a worse prognosis. While earlier studies of antiarrhythmic drug therapy did not demonstrate a survival benefit, more recent evidence suggests that rhythm-control strategies, particularly when initiated early and using catheter ablation, may improve outcomes.

In addition, patients with heart failure are at increased risk of life-threatening ventricular arrhythmias and sudden cardiac death. Prophylactic implantation of an implantable cardioverter-defibrillator (ICD) has been shown to improve survival in selected patients. However, with the emergence of highly effective contemporary heart failure therapies, the indications for preventive ICD implantation are being reconsidered, and current research aims to better personalize ICD therapy. Catheter ablation is also increasingly used for the treatment of ventricular tachycardia in patients with heart failure and has been proposed as a preventive strategy.

Overall, the coexistence of heart failure and arrhythmias represents a major clinical challenge with substantial consequences for patient morbidity and mortality. Continued research is therefore essential to improve risk stratification and treatment strategies, and a broad range of research initiatives addressing these questions are currently ongoing in Sweden.

Why some hearts adapt and others fail: Exploring the biology and variability of the failing heart

Kristjan Karason

Heart failure reflects a spectrum in which the myocardium may either adapt and recover or progress toward irreversible structural disease. Ventricular remodeling is the central determinant of this trajectory, shaped by the interaction between the initial myocardial injury, the biological stress response, and subsequent cellular and tissue remodeling. Diverse insults converge on shared pathways that drive myocyte loss, fibrosis, and changes in ventricular structure. The extent and pattern of myocardial fibrosis emerge as key

determinants of reversibility, representing the anatomical memory of injury. Recovery is more likely when myocardial plasticity is preserved, injury is reversible, and treatment is initiated early. Patient-level factors, comorbidity burden, and therapeutic response further modify outcomes. Understanding why some hearts remodel adaptively while others fail provides a framework for predicting reversibility and guiding treatment strategies in heart failure.

Imaging with echocardiography in CHF

Maria Eriksson

Heart failure (HF) is a clinical syndrome characterized by typical symptoms and signs in conjunction with objective evidence of cardiac dysfunction. Contemporary HF classification increasingly rely on precise HF phenotyping to improve risk stratification and enable individualized therapeutic approaches.

Echocardiography, as the first-line non-invasive imaging modality, permits detailed evaluation of left ventricular (LV) systolic and diastolic function, characterization of LV remodeling patterns, estimation of LV filling pressures, and assessment of right ventricular involvement, pulmonary hypertension, and concomitant valvular heart disease.

Current classification of HF is based on LV ejection fraction (LVEF), distinguishing HF with reduced ejection fraction (HFrEF; ≤40%), HF with mildly reduced ejection fraction (HFmrEF; 41–49%), and HF with preserved ejection fraction (HFpEF; ≥50%). While LVEF remains central to phenotypic categorization, echocardiography is equally essential for the evaluation of diastolic function. Integration of mitral inflow velocities, tissue Doppler–derived mitral annular velocities, left atrial volume index, and tricuspid regurgitation velocity enables estimation of LV filling pressures and grading of diastolic dysfunction. This is particularly relevant in HFpEF, where LVEF is preserved despite elevated filling pressures and overt clinical manifestations. Myocardial deformation imaging - global longitudinal strain, allows detection of subclinical LV systolic dysfunction and provides incremental prognostic information beyond conventional measures. More recently, artificial intelligence–enabled algorithms have been incorporated into echocardiographic workflows, enhancing automated quantification and supporting better identifying HF phenotypes.

This lecture aims to provide an overview of echocardiographic assessment in HF phenotypes, emphasizing the clinical utility, strengths, and limitations of key echocardiographic variables.

Why are biomarkers important in CHF – the evidence and how I use them?

Kieran Docherty

In this lecture, I will explore why circulating biomarkers are central to contemporary heart failure (HF) care, examining both the evidence base and their practical application in clinical decision-making.

I will review the diagnostic and prognostic performance of natriuretic peptides (BNP and NT-proBNP), highlighting their role in ruling out HF, stratifying risk, and prioritising

echocardiography. The evidence supporting high-sensitivity cardiac troponin as a marker of myocardial injury and incremental prognostic risk will also be discussed. Beyond individual tests, I will consider potential multimarker strategies that reflect the complex biology of HF, including biomarkers reflecting fibrosis, congestion and inflammation. Finally, I will outline how we can integrate biomarkers into routine practice to support diagnosis, inform prognosis, guide follow-up intensity and treatment.

Investigation of specific cardiomyopathies and therapeutic implications

Arash Mokhtar

Cardiomyopathies represent a heterogeneous group of myocardial disorders with diverse aetiologies and clinical presentations, and remain an important cause of heart failure and arrhythmias. A structured, phenotype-driven approach is essential to guide diagnostic work-up and management.

This lecture will focus on the clinical approach to patients presenting with a hypertrophic phenotype ("the thick heart"), outlining how to differentiate between underlying causes such as sarcomeric hypertrophic cardiomyopathy, cardiac amyloidosis, and other phenocopies including Fabry disease. Emphasis will be placed on practical clinical assessment, multimodality imaging, and the identification of key diagnostic "red flags" that guide further investigations.

Finally, the therapeutic implications of establishing the correct aetiology will be discussed, including disease-specific treatments and management of heart failure and arrhythmias. The aim is to provide a framework for clinicians to move from phenotype to aetiology and ultimately to targeted treatment.

A pathologist's perspective on pathophysiology in congestive heart failure

Anders Oldfors

Investigation of myocardial tissue using histopathological and molecular biological techniques is essential for diagnostic evaluation and for gaining insight into the pathophysiology of congestive heart failure, especially in genetic and acquired cardiomyopathies.

Although impaired energy metabolism is a common feature of heart failure, endomyocardial biopsy may identify primary defects in carbohydrate metabolism or mitochondrial function within cardiomyocytes as the underlying cause. In addition, genetic disorders of cellular metabolism, such as lysosomal storage diseases, can disrupt cardiomyocyte function through excessive accumulation of undigested substrates, as demonstrated by histopathological findings. In inflammatory and toxic cardiomyopathies, the pathophysiology of congestive heart failure frequently involves extensive myocyte necrosis, although additional toxic and metabolic mechanisms may also contribute.

Regardless of etiology, myocardial fibrosis represents a key component of chronic heart failure and typically follows acute events such as ischemic heart disease and myocarditis. Fibrosis and fatty replacement are also characteristic features of several genetic

cardiomyopathies and chronic inflammatory conditions, including sarcoidosis, leading to progressive loss of cardiomyocytes. In systemic disorders such as amyloidosis, extracellular deposition of amyloid fibrils impairs myocardial contractility and is accompanied by cardiomyocyte damage.

Thus, endomyocardial biopsy provides important added value to other, less invasive, techniques by elucidating disease-specific pathophysiological mechanisms, thereby supporting more targeted therapeutic strategies.

Right heart failure and interplay with left heart failure. Myth or reality?

Marco Astengo

Right ventricular (RV) function is a prognostic marker in patients with heart failure (HF). Given the susceptibility of the RV to afterload, it is believed that pulmonary hypertension plays a major role in the development of RV dysfunction. Indeed, RV afterload might represent the link between the severity of LV failure and the degree of RV dysfunction. We hypothesize that the prognostic importance of RV function derives from its ability to summarize the degree of LV failure and RV afterload.

HF is characterized by episodes of decompensation alternating to periods of clinical stability. The dynamic response of the RV to different haemodynamic conditions suggests that following RV function over time might give insight in the trajectory of LV failure. Consequently, grading RV function could allow to follow both RV and LV performance over time. To date, however, grading RV function is seldom attempted. In order to grade RV function and facilitate re-assessment over time, we have recently proposed a multiparametric RV dyfunction score. The score is based on the pathophysiology of RV dysfunction and is easily obtained from a standard echocardiogram. It ranges from 0 to 5 and takes into account: 1) the systolic pulmonary artery pressure (afterload), 2) the tricuspid annular plane systolic excursion (contractility), 3) the end-diastolic area of the RV (dilation), 4) the degree of tricuspid regurgitation (deformation) and 5) the collapsibility of the vena cava inferior (RV filling pressure).

In patients with new-onset HF, the improvement in LV function and RV afterload after therapy titration was paralleled by an improvement in RV function. In patients with advanced HF, undergoing right heart catheterization and echocardiography, the degree of RV dysfunction was associated to indexes of LV decompensation and to increased RV afterload. Furthermore, grading RV function with the RV dysfunction score improved risk stratification, both in patients with new-onset and advanced HF.

Heart Failure and valve diseases – when is intervention or surgery preferred?

Anders Jeppson

Heart failure secondary to valvular heart disease represents a significant and potentially reversible cause of cardiovascular morbidity and mortality worldwide. Structural abnormalities of the cardiac valves impose chronic pressure and/or volume overload on the myocardium, ultimately leading to ventricular remodeling, systolic and/or diastolic dysfunction, and symptomatic heart failure. In contrast to primary cardiomyopathies,

however, valvular heart disease often constitutes a mechanical problem amenable to definitive correction. Surgical intervention therefore occupies a central role in the management of selected patients, with the potential not only to alleviate symptoms but also to alter the natural history of the disease.

The decision to pursue surgical treatment in patients with heart failure due to valvular disease is guided by symptom burden, valve lesion severity, ventricular function, and overall operative risk. Current guidelines emphasize timely intervention before irreversible myocardial damage occurs. Importantly, the presence of heart failure does not preclude surgery; indeed, in many cases it strengthens the indication, particularly if dysfunction is attributable to the valve lesion itself.

Multidisciplinary heart team discussions, integrating cardiologists, cardiac surgeons, anesthesiologists, and imaging specialists, are now standard practice to individualize treatment decisions. The evolution of transcatheter technologies has expanded therapeutic options, particularly for high-risk patients, while reinforcing the importance of multidisciplinary evaluation. Optimal outcomes depend on precise timing, careful patient selection, and integration of surgical and medical strategies.

Clinical Assessment of Advanced Heart Failure: When to Consider Advanced Therapies

Kristjan Karason

Timely referral of patients with advanced heart failure to specialized centers is critical to optimize access to advanced therapies, yet referral is often delayed in clinical practice. While guideline criteria and prognostic scores help identify high-risk patients, they often recognize advanced disease rather than progression. This presentation emphasizes a trajectory-based clinical approach to referral. Progressive instability despite optimal therapy — reflected by worsening cardiac function, intolerance to guideline-directed medical therapy, recurrent hospitalizations, and emerging multi-organ involvement — should prompt evaluation. Cardiorenal and cardiohepatic interactions further accelerate deterioration. Early referral preserves treatment options and improves outcomes. Recognizing dynamic disease progression, rather than waiting for irreversible decline, is central to optimal timing of referral for advanced heart failure therapies.

Heart Transplantation and LVAD Bridge-to-HTx – why is it considered gold standard?

Göran Dellgren

Severe Heart failure or advanced heart failure is associated with very high mortality. Timing in replacement therapy is essential, and in selected patients mechanical circulatory support and heart transplantation can be great options with good HRQoL and long-term survival.

Surgical intervention with left ventricular assist devices as bridge-to-heart transplantation as well as heart transplantation occupies a central role in the management of selected patients. Indications, surgical management, complications, long-term management and survival will be covered.

Myocardial Recovery: Future Challenges and Opportunities for MCS

Mandeep Mehra

Mechanical unloading with durable mechanical circulatory support has reshaped advanced heart failure from a traditionally irreversible condition into a biologically modifiable state characterized by varying degrees of myocardial remission. Clinical recovery during left ventricular assist device support reflects reverse remodeling across structural, functional, and molecular domains; however, remission remains heterogeneous and seldom represents true biological normalization.

Increasing evidence supports the concept of transcriptional hysteresis, whereby prior myocardial injury imprints persistent genomic and epigenetic memory despite apparent functional recovery, predisposing patients to relapse following device explantation. Accordingly, recovery exists along a continuum ranging from partial remission to sustained myocardial restoration. Outcomes data indicate that although complete responders may achieve durable freedom from recurrent heart failure, partial responders remain susceptible to late deterioration, highlighting the need for mechanistic phenotyping beyond conventional clinical metrics. Integrating longitudinal clinical outcomes with molecular and transcriptional signatures during mechanical unloading offers a translational framework to distinguish remission from cure and to guide strategies aimed at stabilizing recovery and preventing recurrence.

The likelihood of myocardial remission appears greatest in younger patients with shorter heart failure duration and non-ischemic cardiomyopathy, in whom successful decommissioning of durable MCS is achievable and aligns with the broader objective of extending lifespan while compressing morbidity in advanced heart failure.

Bridging the gap between the GP and the cardiologist – better Heart Failure Care Starts in Primary Care

Veronica Milos Nymberg

Milos Nymberg conducts clinical and epidemiological research on prevention and treatment of cardiovascular disease in primary health care, digital care services and health equity. Her research focuses on improved diagnosis and guideline-directed medical therapy for heart failure patients, cross-disciplinary collaboration and prognostic tools such as ECG-based Heart Age and biomarker analyses.

HF patients are best managed by nurses – this is how we do it

Maria Liljeroos

Heart failure (HF) is a lifelong condition requiring continuous management rather than isolated medical interventions. Beyond pharmacological treatment, patients live with a high daily symptom burden and complex self-care demands, highlighting the need for coordinated, person-centred care. Nurses play a central role in HF management, often acting as care coordinators within multidisciplinary teams and providing continuity across the care pathway.

Nurse-led HF care aligns with European Society of Cardiology recommendations emphasizing structured follow-up, patient education, and self-care support. Nurses translate clinical guidelines into everyday practice through repeated and individualized education, involving both patients and informal caregivers to recognize early signs of deterioration and respond appropriately. As patients often meet nurses more frequently than other healthcare professionals, nurses are uniquely positioned to support long-term disease management.

Evidence underscores the importance of sustained self-care support. In a secondary analysis of the COACH-2 study including 167 HF patients (mean age 73 years), declining self-care behaviour was associated with significantly higher all-cause and cardiovascular hospitalisation rates compared with patients maintaining good self-care over time, emphasizing the need for continuous follow-up throughout the HF trajectory.

The early post-discharge period is particularly vulnerable. Findings from STRONG-HF demonstrate that rapid optimization of guideline-directed medical therapy requires frequent monitoring and close follow-up, which nurse-led models facilitate in real-world care through early identification of side effects, adherence support, and collaboration with physicians in medication titration.

Nurse-led HF care does not replace physicians but strengthens multidisciplinary collaboration. Investing in nurse-led continuity, education, and individualized support is essential to improve outcomes in heart failure — because effective HF care happens not only in hospitals, but every day in patients' lives.

What is the evidence for fluid and salt restrictions in CHF patients?

Linn Höög

Fluid and salt restrictions have long been recommended as part of the non pharmacological management of patients with chronic heart failure (CHF). However, the strength of evidence supporting these practices remains uncertain. Research on fluid restriction is limited by small sample sizes, heterogeneous study designs, and inconsistent methodologies. As a result, evidence for its benefit remains inconclusive. Existing randomized controlled trials, as well as systematic reviews and meta analyses, have not demonstrated significant effects on mortality, hospitalizations, NYHA-class, or natriuretic peptide levels. In contrast, fluid restriction has been associated with increased thirst distress and may negatively affect quality of life. Evidence for salt restriction is similarly ambiguous. Systematic reviews and meta analyses have not been able to confirm a clear benefit regarding mortality, cardiovascular outcomes, or hospitalization risk in patients with CHF. Some analyses even suggest that salt restriction may increase the risk of hospitalization and all cause mortality, though no consistent improvement in major clinical outcomes has been demonstrated.

Given these uncertainties, current evidence does not strongly support routine fluid or salt restriction for patients with CHF. The 2024 ESC consensus document accordingly recommends advising patients to maintain a normal intake of fluids and salt. An individualized approach to dietary salt and fluid management, tailored to patient's phenotype, symptoms, and preferences is also emphasized.

Telemonitoring in Advanced Heart Failure – Clinical benefits, personal experience, and ongoing researche

Tomas Rydenstam Mellberg

Telemonitoring in heart failure has evolved from small pilot initiatives to large, randomized trials and real-world implementation as an integral component of contemporary heart failure care. Both non-invasive and invasive telemonitoring strategies have demonstrated clinically meaningful benefits across the heart failure spectrum, including reductions in hospitalizations, improved quality of life, and more effective delivery of guideline-directed medical therapy (GDMT). These effects are particularly relevant in patients with advanced heart failure, who are characterized by a high symptom burden, frequent clinical instability, and substantial healthcare utilization.

Early telemonitoring efforts primarily focused on surveillance and early detection of decompensation. More recent developments have expanded the scope toward proactive optimization of therapy, enabling structured, home-based titration of GDMT supported by remote monitoring of vital signs, laboratory parameters, and patient-reported outcomes. This shift has been supported by growing evidence that telemonitoring can facilitate faster and safer achievement of target doses. In advanced heart failure populations, where rapid clinical deterioration and dose-limiting adverse effects are common, such approaches may facilitate faster yet safer achievement of target doses while reducing the need for frequent in-person visits.

Accumulating data suggests that the clinical impact of telemonitoring is highly dependent on patient selection and timing. The greatest benefits appear to be observed in high-risk patients during vulnerable phases, such as the early post-discharge period following heart failure hospitalization, when the risk of clinical deterioration is highest. In more stable chronic heart failure, telemonitoring may serve a complementary role in long-term surveillance and treatment adherence.

In Sweden, telemonitoring is increasingly being adopted across heart failure clinics, reflecting a transition from research settings to routine clinical practice. Ongoing challenges include defining the optimal duration of monitoring, identifying patients most likely to benefit, and integrating telemonitoring efficiently into existing care pathways. Together, current evidence supports telemonitoring as a scalable tool to improve outcomes and optimize therapy in both heart failure and advanced heart failure populations.

This is how I work up severe heart failure patients – and where we find them?

Oscar Braun

Advanced heart failure is associated with poor prognosis, high symptom burden, and frequent hospitalizations despite optimal guideline-directed medical therapy. In contrast, outcomes after heart transplantation and durable left ventricular assist device (LVAD) therapy are excellent. In Sweden, median survival following heart transplantation now exceeds 20 years, illustrating the substantial survival benefit that can be achieved with timely access to advanced therapies. Despite these advances, many patients with advanced heart failure are either not recognized or are referred to specialized centers at a very late stage of disease. Delayed identification reduces eligibility for transplantation or LVAD therapy due to progressive end-organ dysfunction, frailty, or hemodynamic instability.

Consequently, potentially life-prolonging treatment opportunities may be missed, leading to avoidable morbidity and mortality. It is of great importance to strengthen the collaboration between referring hospitals and specialized advanced heart failure centers to facilitate the identification of potential candidates for advanced therapies.

Increased awareness of referral criteria, dissemination of current evidence, and structured communication pathways between regional hospitals and the advanced heart failure center are critical to ensuring timely evaluation. A proactive and coordinated regional approach may facilitate earlier referral, optimize patient selection, and improve outcomes.

Beyond the Pump: Reframing Cardiogenic Shock in Heart Failure?

Mandeep Mehra

Cardiogenic shock in acute-on-chronic heart failure represents a distinct phenotype best understood as "primed shock", a state in which chronic maladaptation precedes and amplifies acute hemodynamic collapse. Unlike infarct-related shock, which evolves sequentially from abrupt pump failure, primed shock arises from the simultaneous convergence of biventricular dysfunction, systemic venous congestion, microcirculatory impairment, immune activation, endothelial injury, and gut-derived endotoxemia. This preconditioned milieu fosters a feed-forward cascade in which restoration of cardiac output fails to reverse peripheral hypoperfusion, a phenomenon termed "hemodynamic dissonance".

Elevated stressed blood volume, glycocalyx disruption, inducible nitric oxide synthase activation, inflammatory cytokine surge, and lipopolysaccharide translocation collectively sustain organ injury despite macrocirculatory rescue. The frequent emergence of mixed cardiogenic–vasodilatory shock reflects this compounded biology. Recognizing primed shock reframes acute-on-chronic heart failure shock as a multisystem disorder demanding disease-modifying strategies that target congestion, inflammation, endothelial dysfunction, metabolic imbalance, and gut integrity alongside mechanical circulatory support, rather than reliance on pump-centric hemodynamic optimization alone.

Cardiogenic shock in the ICU – how and when should we use ECMO?

Michael Broomé

Veno-arterial ECMO (VA ECMO) has not been shown to increase survival in cardiogenic shock in randomized trials. VA ECMO is not a treatment of a specific disease, but rather a life-support measure aiming to buy time and allow treatment or recovery of the underlying disease state causing the life-threatening circulatory failure. The prognosis of the patient is therefore mainly determined by the success or failure of the disease specific treatment, eg. revascularization in acute myocardial infarctions, anticoagulation in pulmonary embolism, antibiotics in septic shock, spontaneous recovery from stunning and unknown disease mechanisms in myocarditis, Takotsubo cardiomyopathy and post-cardiotomy patients. VA ECMO usually loads the left ventricle and unloads the right ventricle. Optimizing conditions for recovery includes choosing correct cannula

dimensions and placement combined with careful titration of ECMO flow, blood volume, vasoactive and inotropic drugs, sometimes combined with beta-blockers and anti-arrhythmic drugs. ECMO is sometimes combined with intra-aortic ballon pumps and other cardiac mechanical support devices. ECMO in cardiogenic shock should be seen as one tool in a larger toolbox aiming for myocardial recovery, while providing systemic perfusion, oxygen delivery and avoiding systemic and pulmonary congestion. A novel paradigm is presented where "the hemodynamic envelope" and "balanced heart failure" are key concepts emphasizing the importance of right-left ventricular interactions.

Cardiogenic Shock Registry Outcomes – What's New and How to Improve

Maryjane Farr

This talk will survey the genesis of cardiogenic shock (CS) registries in Europe and the United States, and key results over the past two decades including the original SHOCK Trial Registry, IABP-SHOCK II, FAST-MI, Altshock-2-Registry, NCSI, CSWG, CCTN, AHA CS Registry and the SCAI Shock Staging Validation Registries. I will discuss practice changing findings, limitations and burdens of registry data collection and analysis, and new opportunities for identifying patients in CS through automated serial SCAI staging in the Electronic Health Record. Adaptive platform trials embedded into Registries will also be reviewed.

ECPR – A Resource Intensive Treatment: For Whom and When?

Bengt Redfors

After 20 minutes of refractory cardiac arrest, survival with favorable neurological outcome drops below 1%. Extracorporeal cardiopulmonary resuscitation (ECPR) can restore circulation during ongoing CPR, serving as a bridge to cardiac recovery or definitive treatment.

Three randomized trials (ARREST, Prague OHCA, INCEPTION) suggest benefit in selected patients within experienced systems, but results vary, highlighting that ECPR is not a universal rescue strategy. A critical challenge is patient selection. A systematic review of 93 ECPR programmes worldwide revealed substantial heterogeneity in inclusion criteria, with no consensus on age limits, rhythm requirements, or time thresholds.

Furthermore, ECPR demands highly specialised teams and infrastructure, creating geographic inequities where large distances to experienced centres may preclude treatment within a viable time window. Probabilistic prediction models integrating multiple weighted physiological variables obtained during CPR show promise for improving selection beyond traditional binary criteria. This lecture reviews the current evidence and discusses the system requirements for effective ECPR delivery.

*Quality-of-life after ECMO and intensive care for acute heart failure – what's known?**

Inga lára Ingvarsdóttir

The long-term outcome of ECMO treatment is not only described by survival, but also by various measurements of health-related quality of life (HRQoL). The latter has gained increased interest in recent years and is now considered an important part of reporting outcome. It is known that survivors of severe illness in general, requiring treatment in an intensive care unit (ICU), can develop a new onset of symptoms after their critical illness.

These may include cognitive, psychological and physical decline, collectively known as post intensive care syndrome (PICS), that persist after discharge from the acute care and affect quality of life. In addition, symptoms of post-traumatic stress disorder affect up to 25% of patients surviving ICU stay, with indications that the same applies to survivors of ECMO treatment. However, long-term effects on HRQoL are still not as well studied in ECMO patients although evidence suggest that it may be similar to other ICU survivors.

The presentation will focus on long-term outcome after severe illness in the ICU, including what we know about ECMO survivors.

Jöns Jacob Berzelius

Jöns Jacob Berzelius, one of the most prominent natural scientists of the 19th century and one of the seven men who founded The Swedish Society of Medicine.

Jöns Jacob Berzelius, one of the most prominent natural scientists of the 19th century, was born in 1779 in Väversunda, in the county of Östergötland in southern Sweden, a region with rich cultural traditions. Orphaned at an early age, he went to several foster homes and received his schooling in nearby Linköping. After graduating in medicine at the University of Uppsala, he moved to Stockholm, where he became assistant master without pay at the so-called »Surgical School«, and worked as a doctor for the poor. At the age of 28 he became professor of medicine and pharmacy.

In 1808 Berzelius was one of the seven men who founded The Swedish Society of Medicine »For the perfection of science through mutual mediation of knowledge and collective experience, for the promotion of friendly confidence between doctors«.

Berzelius has enriched our knowledge of nature of life phenomena, established the atomic weights of most of the known elements, presented his electrochemical theory for the understanding of the nature of chemical compounds and laid the foundation for the sciences of the chemistry of rock types.

He also found that elements combine with each other according to fixed numerical relationships.

In addition to this, in his striving for order and method, with his talent for simplicity and clarity in expression, he created the chemical symbolic language in 1813, which since that time has been an essential instrument of chemistry.

With time he became a practised lecturer but preferred to express himself in writing. Impressive are the great scientific works where he also demonstrated his interest and ability to spread knowledge about the latest advances of natural sciences.

Parts of this text is found in:
Berzelius – Creator of the chemical language, by Carl Gustaf Bernhard, the Royal Swedish Academy of Sciences

The Swedish Society of Medicine

The Swedish Society of Medicine (SSM) is the independent scientific and professional organisation of the Swedish medical profession.

The Swedish Society of Medicine (SSM) is the independent scientific and professional organisation of the Swedish medical profession. We were founded in 1808 and are one of the oldest medical organisations in Europe. Science, education and quality is the SSM motto and the foundation of SSM activities. We contribute with more than SEK 51 million to medical research every year. The SSM represents the medical profession in various inquiries and consultations. We organise scientific meetings, seminars and debates to highlight medical research in important topical fields.

History of the SSM building
In 1879, the Swedish Society of Medicine moved from what was then the home of Karolinska Institutet at Norr Mälarstrand to its own premises in Jakobsgatan in Stockholm. It soon outgrew this location and a search for new premises was resumed.

On Walpurgis night in 1889, six men were inside the Katarina lift at Slussen in Stockholm. A fault developed in the machinery, causing the lift cage to fall. One of the passengers, Carl Westman, was injured, but a fellow passenger, Johan Rissler, a surgeon and member of the building committee of the Society of Medicine, immediately assisted him.

In 1904, the Society announced an architectural competition for a building on a site it had purchased in Klara Östra Kyrkogata. The winner was Carl Westman, and the building was finished two years later.

The Society's building which dates from 1906, was breakthrough for the architect Carl Westman and the national romantic style architecture he favoured. The building itself is a work of art – from its facade of handmade brick and Christian Eriksson's granite reliefs in the entrance to its mosaic floors, carved balustrades, chandeliers, and ventilation grilles – all Westman signatures. The building today is a Swedish, turn of the century architectural treasure.

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