

Heart Failure: An Exploration of Recent Advances in Research and Treatment (A)

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Supplement Aims and Scope

The supplement aims to provide readers with an exploration of recent advances in research on and treatment of heart failure. This includes but is not limited to the following topics:

- Pathophysiology
- Evaluation
- Management
- Diagnosis
- Prognosis
- Treatment
- Screening
- Prevention
- Epidemiology
- Risk factor modification

- Systematic reviews
- Observational studies
- Commentary on clinical trials
- Risk and safety of medical interventions
- Epidemiology and statistical methods
- Evidence-based medicine
- Evaluation of guidelines
- Translational medicine

Article types include original clinical and basic research articles, case reports, commentaries, meeting reports, methodology, perspective, research proposal, reviews, software/database reviews, and technical advance.

Hear function fails when the organ is unable to pump blood at a rate proportional to the body's need for oxygen or when this function leads to elevated cardiac chamber filling pressures (cardiogenic pulmonary edema). Despite our sophisticated knowledge of heart failure, even so-called ejection fraction-preserved heart failure has high rates of mortality and morbidity. So, novel therapies are sorely needed. With over a million hospitalizations annually—up 175% over the past 25 years—and costs of nearly \$15.4 billion dollars, acute heart failure is a critically-important health concern. Furthermore, half of patients discharged from the hospital are readmitted within half-a-year. In-hospital mortality remains high between 4–7%.^{1,2} Heart failure is a significant

problem as the population ages. The prevalence is 2.5% of the U.S. population or 5 million patients (from the National Health and Nutrition Education Survey: NHANES).²

Common etiological mechanisms of heart failure include coronary ischemia, valvular disease, hypertension, and diastolic dysfunction. Yet, other causes include: post-partum cardiomyopathy, post-infectious, chronic tachycardia, metabolic dysregulation, adverse medication side effects (particularly adriamycin chemotherapy), orphan disease Duchenne's Muscular Dystrophy, infiltrative diseases (such as amyloidosis), and inflammatory/connective tissue diseases (such as systemic lupus erythematosus). When known causes of heart failure are excluded then heart failure is classified as idiopathic. Less often



studied versus chronic heart failure, is acute decompensated heart failure associated with abrupt-onset symptoms associated with hospitalization. Nearly half of admitted patients with heart failure have preserved ejection fraction.^{2,3}

Survival among heart failure patients long-term may be improved with β -blockers,⁴ angiotensin converting enzyme inhibitors,⁵ aldosterone antagonists,⁶ electrophysiology devices such as automatic implantable cardiovascular defibrillators, and vasodilators. Other drugs such as digoxin and diuretics do not alter death rates—digoxin reduces hospitalizations, while diuretics (furosemide or lasix) improve symptoms.

Another aspect of heart failure is diastolic dysfunction with preserved left ventricular ejection fraction, accounting for half of hospitalizations. Pathophysiologically, there is concentric remodeling and increased left ventricular end diastolic pressure from a stiff left ventricle, thereby preventing relaxation. Medications for diastolic dysfunction are similar to systolic dysfunction: ACE inhibitors, ARBs, diuretics, and β -blockers.⁷

Left ventricular assist devices (LVADs) as both a bridge to heart transplantation or as a destination unto itself have been developed. Limitations to LVADs entail gastrointestinal bleeding, pump thrombosis, driveline infection, and late right heart failure and aortic insufficiency. Cardiac transplantations are limited by a relatively small annual donor pool (<4000)⁸ and 60% five-year survival.⁹

Notable manuscripts in this supplement to *Clinical Medicine Insights: Cardiology* “Heart Failure: An Exploration of Recent Advances in Research and Treatment (A)” review viable options for end-stage heart failure. Dr. Allison McLarty discusses mechanical circulatory support and the role of LVADs in Heart Failure Therapy. Dr. McLarty presents a practical review and guideline for LVAD use.⁸ Dr. Fahad Gilani *et al.* discuss “Percutaneous Mechanical Support in Cardiogenic Shock: A Review” and note that mortality is not improved with Intra-Aortic Balloon Pumps (IABP), Extracorporeal Membrane Oxygenation (ECMO) or percutaneous LVADs.¹⁰

Dr. David Bejar and colleagues review sundry abnormal substances which may infiltrate the heart to lead to “Infiltrative Cardiomyopathies” as a prelude to heart failure. While Fabry Disease necessitates enzyme replacement with α -galactosidase A, other diseases like hemochromatosis warrant phlebotomy and deferoxamine, and AL cardiac amyloid may respond to bortezomib, yet other etiologic mechanisms are incurable, leading to heart transplantation.¹¹

An overlooked topic relates to geriatrics, so in regard to heart failure, Drs. Deena Goldwater and Sean Pinney bring up for discussion “Frailty in Advanced Heart Failure: A Consequence of Aging or a Separate Entity?” With the prevalence of frailty higher at 20% of the heart failure population versus 10% in a matched aged cohort, the molecular mechanisms underpinning these differences warrant investigation.¹²

As a complementary article to the mechanical support reviews noted *supra vide*, Dr. Harsh Patel offers recommendations on monitoring for “Complications of Continuous-Flow

Mechanical Circulatory Support Devices” with echocardiography and judicious use of inotropic agents and antibiotics. Dr. Patel points out that AICDs pre-LVAD are kept for their improvement in mortality, however, mortality worsens with AICD shocks.¹³

Finally, I would like to comment on “Reversible Cardiomyopathies” by Dr. Harsh Patel and co-authors, which reminds us to search the treatable, such as Sympathoexcitation-Induced Takotsubo Cardiomyopathy due to excess catecholamines from autonomic dysfunction. Despite spontaneous reversibility aided with beta-blockers and angiotensin converting enzyme inhibitors, heart recidivism rates are 11.4% at 4 years.¹⁴

We are fortunate to be practicing at an exciting time for the profession of medicine—an era of rapid evolution in technology and knowledge-base in basic science. What this may hold for the short-term treatment of heart failure remains to be seen, but ultimately the goal will be to help us improve upon current practice. Bioinformatics, computational biology, genomics, proteomics, metabolomics, pharmacology, and quantitative epidemiology may be part-and-parcel of the next issue of “Heart Failure: An Exploration of Recent Advances in Research and Treatment.”

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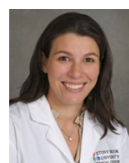
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