CardioVascular Learning Network

Recognizing & Treatment Options in Patients with **Heart Failure with Preserved Ejection Fraction (HFpEF)**

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This program is intended for physicians, cath lab teams and institutions treating heart failure patients, interested in learning more about future heart failure treatment technologies.

LEARNING OBJECTIVES

- Understand the definition and prevalence of patients with Heart Failure with preserved Ejection Fraction (HFpEF)
- Learn multiple strategies to diagnose HFpEF
- Identify ways to find HFpEF patients in the healthcare system, including the HFpEF score
- Recognize the importance of a multi-specialty team approach to HFpEF

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FACULTY

The faculty have reported the following:

Dr. Colaco: Faculty/Presenter - Corvia Medical

Dr. Hibbert: has disclosed no relevant financial relationship with any ineligible company (commercial interest).

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Planning for this activity was conducted by faculty advisors. To further support the development of the content, additional information regarding professional roles, responsibilities, and teamwork was gathered through survey input from our target audience and/or a comprehensive literature review.

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1. INTRODUCTION

The clinical understanding and approach to heart failure with preserved ejection fraction (HFpEF) is a rapidly evolving area of cardiovascular medicine—a progression that is both necessary and timely given the rising incidence of HFpEF and the growing recognition of its global burden.[1] HFpEF constitutes the majority of heart failure diagnoses and carries a mortality risk similar to heart failure with reduced ejection fraction (HFrEF).[2] In addition to its impact on survival, HFpEF is a major contributor to functional limitation and diminished quality of life for affected patients.

In contrast to HFrEF—which has a well-established therapeutic framework consisting of a range of morbidity- and mortality-reducing medications, approved and investigational device therapies, and durable advanced options such as left ventricular assist devices and heart transplantation—HFpEF has long lacked targeted options. Management has historically focused on symptomatic relief via diuretics, hypertensives, and comorbidity management.

However, this paradigm is shifting. Two pharmacologic classes—SGLT2 inhibitors and non-steroidal mineralocorticoid receptor antagonists—have now demonstrated clinical outcome benefits in HFpEF, marking transformative development in the field. Emerging data also support a role for incretin-based therapies, and numerous ongoing trials are investigating novel molecular pathways and device-based interventions, such as those targeting left-sided filling pressures during exertion to mitigate symptom burden.

To fully capitalize on these advances, clinicians must first recognize HFpEF when it presents, and second, understand how to match individual patients to both established and emerging therapies.

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2. UNDERSTANDING HFpEF

Unlike HFrEF, which is generally a cardiocentric disease process, HFpEF is best understood as part of a broader systemic disease syndrome, where cardiac dysfunction coexists with comorbidities such as obesity, diabetes, and chronic kidney disease. These comorbidities result in a heightened systemic inflammatory state, ultimately leading to the development of excess extracellular matrix or fibrosis within the myocardium, which leads to reduced myocardial compliance, or the heart's diminished ability to relax and fill properly during diastole.

Initially, this impaired compliance manifests as a disproportionate rise in left-sided filling pressures during exertion, when the heart must accommodate 3–5 times normal cardiac output. This leads to classic HFpEF symptoms, particularly exertional dyspnea, often noticeable with stairs, inclines, or moderate physical activity. In the early stages, echocardiographic findings may be subtle or absent, and resting hemodynamics may appear normal. As the disease progresses, patients may develop more overt signs of congestion at rest, such as elevated jugular venous pressure and lower extremity edema. The gold standard hemodynamic definition of HFpEF is a pulmonary artery wedge pressure (PAWP) >15 mm Hg at rest or >25 mm Hg with peak exercise.

In 2021, a universal definition of heart failure was introduced, motivated in part by growing recognition that traditional diagnostic frameworks were inadequate for capturing the full HFpEF spectrum. Existing non-invasive definitions often relied on complex echocardiographic indices of diastolic dysfunction or structural heart changes, which many patients with HFpEF, particularly those with exercise-only abnormalities, do not exhibit. These patients often presented with "unexplained dyspnea", and further confounding factors such as low natriuretic peptides in obesity contributed to underdiagnosis.

The new universal definition characterizes heart failure as the presence of typical signs and/or symptoms caused by structural and/or functional cardiac abnormalities, confirmed by either elevated natriuretic peptides or objective evidence of pulmonary or systemic congestion at rest or with provocation. Heart failure is further characterized by left ventricular ejection fraction, with HFrEF defined as <40%, HF with moderately reduced EF 40%-50%, and HFpEF defined as an ejection fraction >50% (excluding specific cardiac etiologies such as amyloidosis or hypertrophic cardiomyopathy).

Critically, this definition acknowledges that routine diagnostic tools—biomarkers, resting echocardiogram, and right heart catheterization—may be entirely normal in early HFpEF. As such, advanced testing modalities such as diastolic stress echocardiography or invasive exercise hemodynamics may be necessary to establish the diagnosis.

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3. CHALLENGES IN DIAGNOSING HFpEF

The prevalence of HFpEF continues to rise, driven in part by evolving definitions that emphasize functional rather than purely structural abnormalities.[2, 3] Despite this shift and growing interest in novel treatment strategies, HFpEF remains underrecognized in routine clinical practice.3 Exercise-induced HFpEF—defined as a normal resting PAWP with a rise to >25 mm Hg with maximal exercise—is particularly challenging to diagnose. A high level of clinical awareness is essential, as standard testing is often non-revealing (aside from potentially elevated NT-proBNP), and advanced testing at a specialized center is often required to unmask the cardiac limitation.

Notably, HFpEF shows a strong female predominance.[4] Obesity, a well-established contributor to myocardial fibrosis, is an especially potent risk factor for HFpEF in women—more so than in men.[4] However, symptoms such as dyspnea and fatigue are non-specific and frequently misattributed to obesity, deconditioning, or aging, which can delay recognition of a treatable diagnosis.

These symptoms are non-specific and can result from a wide array of underlying conditions. Edema may result from hepatic or renal dysfunction, or be a side effect of commonly prescribed antihypertensives, particularly calcium channel blockers. Dyspnea may reflect an anginal equivalent or signal other cardiopulmonary diseases such as valvular heart disease or chronic obstructive pulmonary disease (COPD). Systemic contributors such as anemia or hypothyroidism must also be considered. Given this overlap, most patients warrant a baseline workup that includes routine bloodwork, ischemic evaluation (e.g., stress testing, CT coronary angiography, or catheterization), and an appropriate pulmonary assessment (CT chest, pulmonary function tests). Deconditioning may also be considered, though it is essential not to anchor on this diagnosis without objectively excluding HFpEF.

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4. IDENTIFYING PATIENTS IN THE HEALTHCARE SYSTEM

While clinical diagnosis is difficult, HFpEF is also frequently missed because of how patients initially interact with the healthcare system. Many individuals with HFpEF initially present with exertional dyspnea or fatigue, symptoms commonly attributed to more familiar conditions such as ischemic heart disease or obstructive lung disease. As a result, they are frequently routed through CAD or pulmonary clinics, where their symptoms may be misattributed—delaying accurate diagnosis and treatment. Compounding the issue, these patients often have normal or near-normal left ventricular EF on echocardiography, which can further obscure the diagnosis unless HFpEF is specifically considered.

Potential patients with HFpEF often enter the healthcare system through a variety of avenues, including primary care, cardiology, pulmonology, geriatrics, and procedural clinics. Because of this broad access pattern, it is critical that providers across specialties maintain a high index of suspicion for HFpEF, particularly in older adults, women, and individuals with comorbidities such as hypertension, diabetes, atrial fibrillation, or obesity.

The complexity of diagnosing HFpEF is further complicated in patients with concurrent cardiac conditions such as aortic stenosis and atrial fibrillation (AF), both of which are prevalent in HFpEF populations. These conditions share similar demographics and clinical presentations (e.g., exertional dyspnea), and although diagnostic and treatment pathways exist for AS and AF, persistent symptoms after successful intervention should prompt thorough evaluation for HFpEF. While diagnostic tools like natriuretic peptides and echocardiographic diastolic parameters may offer clues, they cannot replace invasive hemodynamic testing in patients with a high pretest probability.

Even when comorbid conditions like AS or AF are correctly identified and managed, residual symptoms may persist, highlighting the need to consider HFpEF more broadly. Even when structural or arrhythmic pathology is addressed, clinicians may miss the broader clinical picture. A comprehensive, symptom-based approach is needed to avoid misattribution and identify HFpEF as the true cause of functional limitation.

Post-procedural patients—especially those who have undergone transcatheter aortic valve replacement (TAVR), AF ablation, or left atrial appendage occlusion (LAAO)—warrant particular attention. These interventions often improve structural or rhythm-based metrics but do not address underlying HFpEF. For instance, after TAVR, improved hemodynamics may not fully resolve dyspnea if HFpEF is present, and similarly, AF ablation may stabilize rhythm without addressing diastolic dysfunction. When patients continue to report dyspnea following technically successful procedures, clinicians should routinely consider HFpEF. Use of clinical scoring systems such as the H_2FPEF score or the HFA-PEFF algorithm can guide further investigation and referral, which are detailed further in Section 5.

Ultimately, referral to HF specialists for advanced diagnostics (including stress echocardiography or invasive hemodynamics) should be considered early in the evaluation process. This approach is essential not only to confirm the diagnosis but to match patients with emerging treatment options. A proactive, multidisciplinary approach improves diagnostic accuracy and optimizes outcomes in this complex and frequently misidentified patient population.

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5. DIAGNOSTIC TOOLS AND STRATEGIES

In addition to streamlining the HFpEF diagnostic criteria in recognition of the ongoing diagnostic challenges, another important tool intended to identify at-risk patients is the H_2FPEF prediction score, developed with the specific intent of distinguishing HFpEF from non-cardiac dyspnea in symptomatic individuals.[5] The score has been validated in individuals who meet invasive hemodynamic criteria for HFpEF (both at rest or with exercise), and the key risk factors identified include obesity, hypertension, AF, age >60, pulmonary hypertension, and echocardiographic measures of elevated filling pressures. A major utility of the risk score is in identifying (via an intermediate or high-risk score) those individuals among the unexplained dyspnea population who would benefit from further evaluation with provocative cardiac testing—such as diastolic stress echocardiography or invasive exercise hemodynamics—which assesses cardiac function under exertional conditions to reveal abnormalities not seen at rest.

While invasive hemodynamics remain the gold standard for diagnosis of HFpEF, non-invasive alternatives are increasingly used as an intermediate step. Hemodynamic (rather than ischemic) stress echocardiography may be more acceptable to patients and still provide key physiological insights. The primary diagnostic markers include the E/E' ratio (to estimate left atrial filling pressures) and pulmonary artery systolic pressure, which can reflect exercise-induced pulmonary hypertension, a hallmark of exercise-HFpEF.

That said, the diagnostic performance of diastolic stress echocardiography is variable. Comparisons with invasive testing suggest that E/E' is most useful at the extremes,[6, 7] or when its negative predictive value is applied. This imaging modality continues to evolve with the addition of parameters such as strain imaging and may be considered a reasonable alternative to invasive testing, particularly when interpreted cautiously in the appropriate clinical context.

As an intermediate diagnostic step, invasive hemodynamic testing is resource-intensive, yet offers unparalleled diagnostic clarity, especially in patients with unexplained exertional dyspnea. Many patients are eager for definitive answers after years of being told their symptoms stem from non-cardiac causes such as obesity or deconditioning. In addition to direct PAWP measurements during rest and peak exercise, the PAWP/CO cardiac output (CO) slope can be calculated—where a slope >2 mm Hg/L/min suggests a disproportionate rise in filling pressure consistent with HFpEF.

By simultaneously measuring right atrial pressure, clinicians can also assess the transcardiac pressure gradient during exertion, which is particularly relevant when evaluating patients for investigational therapies such as atrial shunt device trials.

As with all high-resolution diagnostics, technical quality is critical. Key best practices include:

- Using a larger pulmonary artery catheter for signal stability,
- Securing the pressure transducer off the procedural field,
- Applying a heme column in the catheter setup for gentle waveform dampening, and
- Validating PAWP accuracy with wedge saturation >90% when in doubt.

Finally, supine bike exercise, though widely used in most catheterization labs, may not fully replicate symptom onset in all patients—an important limitation to keep in mind when interpreting negative or equivocal findings.

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6. COLLABORATIVE APPROACH TO CARE

The evolving landscape of cardiovascular medicine has fostered growing collaboration between interventional cardiologists and HF specialists. No cardiology subspecialty has seen greater transformation than the treatment of valvular heart disease, driven by the advent of transcatheter therapies. Procedures such as TAVR and transcatheter mitral valve repair/replacement (e.g., MitraClip or TMVR) have expanded treatment options for patients with valvular disease and heart failure. While these procedures are performed by interventional cardiologists, HF specialists play a central role in pre-procedural optimization, patient selection, and post-procedural management, particularly in patients with advanced myocardial disease. The success of this interdisciplinary approach offers a valuable blueprint for other emerging technologies, especially in the context of HFpEF.

The diagnosis and treatment of HFpEF represent a new frontier where collaborative care is increasingly critical, particularly as diagnostic tools like invasive exercise hemodynamics gain prominence. Many patients with HFpEF initially present to interventional cardiology clinics with exertional dyspnea attributed to CAD or valvular pathology, leading to potential misdiagnosis. As awareness grows, interventional cardiologists are playing a greater role in identifying these patients.

Exercise hemodynamics—considered the gold standard for diagnosing HFpEF by detecting elevated filling pressures during stress—is both resource-intensive and underutilized. Its use often exceeds the capacity of current referral systems and trained providers. Therefore, it is imperative that both HF specialists and interventional cardiologists become proficient in ordering, performing, and interpreting these tests to improve diagnostic efficiency and guide therapies, whether through device-based interventions or targeted pharmacologic therapies.

As HFpEF recognition grows, so too does the clinical demand for timely diagnosis and tailored treatment, underscoring the need for formalized collaboration. The overlap between HF and interventional cardiology is now central to optimal care delivery in this complex patient population. The formation of dedicated HFpEF teams or heart failure "heart teams"—similar to those already established in structural heart programs—can improve patient selection, reduce diagnostic delays, and facilitate access to advanced therapies.

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7. CONNECTING PATIENTS TO TREATMENTS

Significant progress has been made in the medical management of HFpEF, with therapies such as sodium-glucose cotransporter-2 inhibitors (SGLT2i), mineralocorticoid receptor antagonists, and angiotensin receptor-neprilysin inhibitors demonstrating efficacy in improving clinical outcomes. These advancements have equipped clinicians with important tools to address the multifactorial pathophysiology of HFpEF. However, despite optimized medical therapy, a substantial proportion of patients continue to experience persistent dyspnea, exercise intolerance, and reduced quality of life. Contributing factors include heterogeneous disease mechanisms, high comorbidity burden (e.g., AF, obesity), and limited targeted therapies for diastolic dysfunction—all of which underscore the unmet need for novel symptom-directed interventions.

In this context, transcatheter and device-based interventions are emerging as promising options for patients who remain symptomatic despite guideline-directed medical therapy. Investigational approaches include neuromodulation techniques such as baroreceptor activation therapy and vagal nerve stimulation, which are designed to modulate autonomic tone and exertional tolerance. Additionally, left atrial decompression via interatrial shunt devices is gaining traction in clinical trials as a strategy to reduce left-sided filling pressures and alleviate dyspnea. Implantable hemodynamic monitoring systems, such as CardioMEMS, provide real-time pulmonary artery pressure data to guide therapy adjustments and reduce decompensation. While these therapies remain investigational and are currently limited to select patients, they represent a critical avenue for those with refractory HFpEF.

Given this rapidly evolving landscape, it is critical to refer patients with HFpEF with persistent symptoms to HF centers of excellence, both to expand access to emerging therapies and to contribute to the evidence base guiding future care. Specialized centers are uniquely equipped to offer advanced diagnostics, clinical trial enrollment, and multidisciplinary collaboration between heart failure specialists, interventional cardiologists, and electrophysiologists. These teams can develop individualized care plans, often investigational therapies where appropriate.

For highly symptomatic patients, such referrals not only offer access to innovative devices, but also represent an opportunity to improve daily function and quality of life. Importantly, they also help clinicians stay engaged with cutting edge HFpEF research and clinical innovation.

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8. CONCLUSION

The diagnosis and management of HFpEF, with its growing prevalence, remains challenging due to its symptom overlap with a wide range of cardiac and non-cardiac conditions. A high index of suspicion of HFpEF is critical, particularly in patients with exertional dyspnea and multiple comorbidities. When clinical suspicion for HFpEF is high based on symptoms and risk factors, invasive hemodynamic assessment remains the gold standard for confirming the diagnosis.

Despite recent advances in medical therapy, a significant proportion of patients continue to experience symptoms and impaired quality of life. In these cases, a multidisciplinary approach is essential. Device-based interventions, advanced diagnostics, and referral to heart failure centers of excellence represent important next steps for patients with persistent symptoms.

As understanding of HFpEF expands, so too must clinical strategies. Providers must stay informed about evolving diagnostic tools, phenotype-specific therapies, and referral pathways. Timely recognition, diagnostic precision, and collaborative care remain the cornerstones of improved outcomes in this complex patient population.

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