



Is your patient HuFFing and PuEFing?

HFPEF: NEW NAME, OLD PROBLEM.

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The facts

Heart Failure with Preserved Ejection Fraction (HFPEF) used to be known as Diastolic heart failure (DHF). DHF (or diastolic dysfunction) refers to abnormalities of left ventricular relaxation seen on echocardiography. The problem was that these abnormalities were often also seen in patients with co-existing Systolic Heart Failure (SHF). Therefore heart failure was “re-divided” into HFPEF and HFREF (Heart Failure with Reduced Ejection Fraction i.e. systolic dysfunction).

It is not universally agreed that they are two separate pathophysiologies, although differing patterns of LV remodelling and disparate responses to medical therapy suggest that they may well be. The exact ejection fraction that divides these two groups is contentious. Most guidelines suggest that HFPEF includes: symptoms and/or signs of heart failure, a non-dilated heart, LVEF>50% and evidence of diastolic dysfunction. Trials have often included patients with an LVEF>40% into this group.

This evidence of diastolic dysfunction is normally uncovered with echocardiography, with features such as: left ventricular hypertrophy, left atrial dilation and other more advanced features such as changes in mitral inflow (E/A ratio, E/E' ratio) and changes in pulmonary vein flow.

Other evidence may include elevated levels of natriuretic peptides and (via invasive measures) elevated levels of left ventricular end-diastolic pressure (LVEDP) or pulmonary wedge pressure (PWP).

Diagnosis

In practice, however, it is often difficult to accurately diagnose in the outpatient setting. The patient is often moderately but non-specifically breathless, with lung function tests and chest x-rays being essentially normal. An echo is often performed, which can again show non specific findings of mild LVH, a dilated left atrium etc. HFPEF is often only finally diagnosed when the patient presents with pulmonary oedema, and the chest x-ray shows the typical features of this.

How to treat?

Treatment of HFPEF is difficult. No specific treatment has been shown to categorically improve the prognosis of this condition. ACE-inhibitors, Angiotensin II receptor antagonists and beta-blockers have all failed to show a convincing impact on survival. There is some data to suggest that statin treatment may be beneficial. Most clinicians treat the fluid overload symptoms of HFPEF with diuretics. Beta-blockers are often used to slow the heart rate and allow more time in diastole for the left ventricle to fill, in an effort to reduce exertional dyspnoea.