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'Diastolic heart failure'

EDITORIAL

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New understanding of heart failure with preserved ejection fraction raises hopes of better treatment.

It is many years since the only factor that was considered pertinent to the assessment and treatment of patients with symptoms of heart failure was systolic contraction of the left ventricle expressed as the ejection fraction.

In the 1980s, Liv Hatle et al. described different diastolic filling patterns through the mitral valve (1). Via simultaneous measurement of intracardiac pressures and gradients, they explained the haemodynamic basis of the signal recorded with Doppler echocardiography. Hatle et al. characterised the normal filling pattern and described restrictive filling, which is common in cases of systolic heart failure. They also showed that patients with symptoms of heart failure and apparently normal systolic function often had diastolic relaxation disturbance (1).

For the past 20 years, description of the diastolic filling pattern has formed part of a comprehensive echocardiographic examination. Assessment of diastolic function has gradually been supplemented with measurement of pulmonary venous flow by Doppler echocardiography, colour Doppler imaging of left ventricular filling, and tissue Doppler imaging.

The importance of diastole has become increasingly apparent over the years, and has helped to explain why patients with apparently normal cardiac function based on their ejection fraction may also experience symptoms of heart failure. Equally important, the new techniques have made it possible to

diagnose and explain different types of heart failure, also for those of us who work at smaller hospitals, far from major research centres and without access to invasive tests.

In international and national guidelines, the term 'heart failure with preserved ejection fraction' is used in place of the less precise 'diastolic heart failure' (2, 3). Similarly, 'heart failure with reduced ejection fraction' is used instead of 'systolic heart failure'. Most patients with 'systolic heart failure' also have diastolic dysfunction, and signs of abnormal systolic function may be detected in patients with 'diastolic heart failure'. The term 'heart failure with mid-range ejection fraction' has been adopted for patients with ejection fraction in the 'grey zone' of 40–49 % (3). This is sensible, especially as it is not always easy to determine whether dysfunction is predominantly diastolic or systolic.

The drugs used to treat heart failure with reduced ejection fraction have well established efficacy (3), and all dampen the neurohumoral activation characteristic of this disorder. To date, there are no treatments with established efficacy for patients with heart failure with preserved ejection fraction (3). We have had to make do with treating underlying factors, such as hypertension, and regulating the heart rate to prolong diastole in the hope of achieving improved filling. Diuretics have good symptomatic efficacy in many patients, but whether they improve prognosis remains unclear. ACE inhibitors have also been recommended on the assumption that they may reduce myocardial hypertrophy and fibrosis and thereby improve diastolic relaxation, in addition to having antihypertensive properties.

Patients with heart failure with preserved ejection fraction appear to be a different population from patients with reduced ejection fraction. They are generally older, more often women, often have hypertension and overweight or other features of so-called metabolic syndrome, renal failure, obstructive pulmonary disease and sleep apnoea (3). By contrast, heart failure in these patients is less often due to coronary artery disease or myocardial infarction. This heterogeneity with a high degree of comorbidity, plus the absence of effective treatments, makes management of this patient group a major challenge.

Amund Treu Røe et al. present a thorough update of recent research on causal mechanisms of heart failure with preserved ejection fraction, based on an extensive review of the literature (4). The authors conclude that a broader perspective is required: rather than regarding heart failure with preserved ejection fraction as an isolated cardiac disorder, it is more appropriate to view it as a clinical syndrome comprising many aetiological subgroups with common pathophysiological features.

Heart failure with preserved ejection fraction is now regarded as a multiorgan syndrome driven by inflammation and endothelial dysfunction. Systemic inflammation leads to endothelial inflammation and microvascular angiopathy, the formation of harmful mediators and reduced bioavailability of substances such as nitrogen monoxide. The consequences may include an impaired vasodilator response, increased arterial stiffness, left ventricular hypertrophy, extracellular fibrosis and increased cardiomyocyte stiffness. This cascade of

pathological changes in the circulatory system helps to explain the symptoms, and also the reduced diastolic filling, which had previously been considered central to the syndrome.

The treatment of underlying factors and comorbid conditions has long been recommended, based more on the belief that this is beneficial for patients with heart failure than on any firm evidence. It is to be anticipated that the scientific justification for such treatment will increase – and that treatment will become more targeted. Increased understanding of the mechanisms underlying symptoms also paves the way for potential new treatments, as described in the article. When such treatments are ready for clinical use, we must hope that good quality studies show them to be beneficial.

The authors conclude with some words of wisdom about the long road ahead. While it is impossible not to agree, there are also strong grounds to believe in continued therapeutic progress!

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