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BMJ 2005;331:415-416
doi:10.1136/bmj.331.7514.415

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Cardiac impairment or heart failure?

“Heart failure” confuses doctors and patients and needs renaming

“There is no disease that you either have or don’t have—except perhaps sudden death or rabies. All other diseases you either have a little or a lot of,” said Geoffrey Rose.¹ This is true of “heart failure”—everybody can have a bit if they try hard enough, by physical exertion or even by emotional shock.² But, apart from transient induced cardiac overload, the term can be used to mean anything from asymptomatic systolic dysfunction to imminent death from pulmonary oedema. Because of widely varying definitions, the epidemiology of heart failure can become almost uninterpretable, with estimates of its prevalence in the United Kingdom

varying from 500 000 to 3 million.³ Moreover, qualitative studies show that many patients are never told that they have heart failure because doctors are understandably reluctant to use the term.⁴ When a label confuses doctors and impairs communication with patients, it seems sensible to change the label.

The recent increase in interest in heart failure began with interventional studies among highly selected patients. They were mainly men aged 60-65 on average, with a history of myocardial infarction or cardiomyopathy and a left systolic ejection fraction of less than 30-35% as measured by cardiac catheterisation or radionuclide ventriculography. After initial success in

BMJ 2005;331:415-6

treating such patients with angiotensin converting enzyme inhibitors, a series of other drugs were tried, usually by addition and using similar selection criteria. These trials have left us a valuable legacy of evidence on the best ways to slow the progression of systolic heart failure in younger men, mainly because of ischaemia. However, most patients with a syndrome of heart failure are not like this.⁵ Their average age in general practice in the UK is 77,⁶ and they mostly have considerable comorbidity. The proportions of women and men even out with age, as do the proportions with and without systolic dysfunction.

To identify patients with heart failure who correspond to the group for which we have an evidence base, clinicians and service providers have focused on improving access to echocardiography. But echocardiography alone cannot diagnose heart failure: it is not the "gold standard." None of the early and important interventional trials used echocardiography to measure systolic ejection fraction because, although it is relatively cheap and accessible, several other methods are more accurate. Echocardiography is an essential tool in assessing the status and severity of heart failure and provides a wealth of structural and dynamic information. But heart failure remains a clinical diagnosis, and functional status and prognosis bear little relation to the ejection fraction alone. In a recent European study researchers, like many clinicians, used an ejection fraction of 50% to define "systolic dysfunction." They found no difference in 10 year survival among patients with ejection fractions above and below this level.⁷ Similar outcomes have also been reported in hospital patients in the UK, using ejection fraction of 40% as the cut-off point.⁸

Heart failure is found chiefly in elderly people who may or may not have impairment of systolic function that is measurable by echocardiography. As every clinician knows, such patients often go in and out of overt heart failure. Because heart failure is a continuum, its definition should be based on the best marker for prognosis. There is little doubt that the best single marker is the level of the cardiac hormone, B-type natriuretic peptide, in blood: measured on a single occasion, it outperforms all other tests, including the systolic ejection fraction and more comprehensive echocardiographic measures, such as the Tei index.⁹ Measuring B-type natriuretic peptide sequentially to determine average serum concentrations or the rate of their change will probably prove even more predictive because this hormone responds quickly to changes in cardiac load. Moreover, evidence is increasing that this hormone could be a much needed marker of response to treatment for heart failure in individual patients.

It is hard to overstate the value of a simple, repeatable, and highly predictive blood test in guiding the treatment of heart failure. Such a test could give patients with heart failure access to the kind of chronic disease management that works successfully in primary care for diabetes and for secondary prevention in coronary heart disease. But before this can become a reality, we need more long term studies of B-type natriuretic peptide as a predictor of response to treatment: several are in progress.¹⁰ Also, by routinely measuring this hormone in patients at risk of heart

failure—those with ischaemic heart disease, high blood pressure, and diabetes—we may be able to prevent or delay the onset of symptomatic heart failure in many of such patients.

B-type natriuretic peptide is a reliable indicator of a struggling heart. Anything which strains or inflames either of the cardiac ventricles increases its serum concentrations, and conversely those levels are not elevated in the absence of ventricular strain or inflammation. In individual patients, further investigation may need to be done to determine the cause of that struggle, but the more we use B-type natriuretic peptide in clinical management, the more we will tend to redefine our concept of "heart failure" and may begin to wonder whether this a helpful label at all.

For doctors, "heart failure" covers a confusingly wide spectrum of illness, whereas for patients it has a deadly ring of finality.¹¹ Failure means the end of hope, and many patients who have been told they have heart failure prefer not to remember the term or let it dominate their lives. This partial denial may have damaging consequences, both psychologically and in terms of adherence to treatment.¹² Given that we already have so much trouble deciding on a definition of heart failure ourselves, it might be kinder, and more accurate, to start calling it cardiac impairment.

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Competing interests: None declared.

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