Heart failure: are we neglecting the silent majority?

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This editorial refers to 'Prognosis of all-cause heart failure and borderline left ventricular systolic dysfunction: 5 year mortality follow-up of the Echocardiographic Heart of England Screening Study (ECHOES)'† by F.D.R. Hobbs et al., on page 1128.

In the last decade, seminal studies from Europe, the USA, and Australasia have defined the epidemiology of heart failure and left ventricular systolic dysfunction in the population.1–3 One of those studies, the aptly named Echocardiographic Heart of England Screening Study (ECHOES), enrolled over 6000 individuals ≥40 years, living in the West Midlands region of England.4 Hobbs and Colleagues provide further important and incremental insights into the epidemiology of heart failure from their study.

First, they report the number of cases of heart failure with a low left ventricular ejection fraction (LVEF) (LVEF <0.40; n = 219) and ‘preserved’ LVEF (>0.40, n = 230), highlighting, as others have done, that approximately half of patients with the clinical syndrome of heart failure do not have a low LVEF.1–3,5 Hobbs et al., however, go one step beyond prior studies in better defining the heterogeneous reality of heart failure. They describe the prevalence of heart failure related to valve disease (n = 97) and atrial fibrillation (n = 133), as well as multiple causes (n = 43; all had a low LVEF). We have been lacking these crucial epidemiological data.

Secondly, Hobbs et al. also describe the number of individuals with a low LVEF who did not have symptomatic heart failure (n = 109). This finding that approximately one-third of patients with a low LVEF do not have symptoms confirms prior reports and re-awakens questions about detection and treatment of these individuals, reinforced by knowledge on their prognosis described below.1–3,6,7

Thirdly, and most importantly, the authors describe long-term prognosis in all these groups of individuals, with some striking findings. Heart failure, irrespective of LVEF, is associated with a greatly reduced survival. Conversely, the risk of premature death is also increased in individuals with a low LVEF, irrespective of whether they have symptomatic heart failure. Specifically, the 5-year mortality rate was 47% in subjects with heart failure and a low LVEF, 38% in those with heart failure and preserved LVEF, and 31% in individuals with a low LVEF without heart failure, but only 7% in the overall population. Crucial new information is shown in Figure 5 of the report from Hobbs et al. Subjects with heart failure and atrial fibrillation, large in number, had a prognosis as poor as individuals with heart failure and a low LVEF. The same was true for individuals with heart failure and valve disease. Patients with heart failure and more than one of these conditions had an even worse prognosis, although the numbers of individuals in these latter two groups were small.

Equally important is the authors’ demonstration that individuals with an LVEF in the range 0.40–0.50 (n = 386) also have a considerably reduced survival when compared with those with an LVEF >0.50 (n = 5447), although not as much as individuals with an LVEF <0.40 (n = 328). This is an important confirmation that ‘left ventricular systolic dysfunction’ is not an ‘all or none’ phenomenon and that there is no magical dividing line between ‘normal’ and ‘abnormal’.5,7 Although the exact numbers are not provided, these data from Hobbs et al. tell us that there are many premature deaths in the population among asymptomatic individuals with either a clearly reduced (<0.40) or borderline (0.40–0.50) LVEF. This silent majority deserves more attention. Who are they? Unfortunately, the authors do not describe their clinical characteristics. What is their natural history? Do these subjects progress to symptomatic heart failure? If so, at what rate and what predicts progression? What are the causes of premature death? Can we better risk stratify these individuals, for example, by measuring blood natriuretic peptides or, perhaps, markers of cardiac electrical instability such as microvolt T-wave alternans? Obviously, the issue underlying these questions is whether progression to the symptomatic state and death can be postponed or avoided.

The broader issue highlighted by this study is how the majority of patients with heart failure or borderline/reduced LVEF have been excluded from clinical trials, which have, until recently, almost exclusively focused on those with symptoms and an LVEF <0.35. At last, we have begun to tackle the problem of heart failure and preserved LVEF.5 Recent studies with renin-angiotensin system blockers in these patients have been encouraging but not definitive; two additional studies are in progress with an angiotensin receptor blocker (I-PRESERVE) and an aldosterone-antagonist (TOPCAT).6,9 In contrast, we have done little to investigate the treatment of patients with a low LVEF but without symptomatic heart failure. Other than the prevention arm of the Studies of Left Ventricular Dysfunction, which compared enalapril with placebo, no large-scale trials have been carried out in these patients.10 Might a beta-blocker be of value in these under-studied groups of patients?

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patients? Are there other treatments that might improve outcome? We should no longer neglect the majority of patients with heart failure, particularly the silent majority.

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References


Clinical vignette

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Unstable single coronary artery

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A 78-year-old male presented for elective cardiac catheterisation following a 1-week history of crescendo angina on a background of longstanding hypertension, hypercholesterolaemia, and transient ischaemic attacks.

Cardiac catheterisation revealed a single coronary artery (SCA) anomaly, with the right coronary artery (RCA) arising from the right coronary sinus continuing on to sequentially form the anatomical circumflex (CX) and the left anterior descending (LAD) artery. A 90% lesion was apparent in the anatomical mid-CX coronary artery isolating the LAD, which filled slowly antegradely. No left main coronary artery was discernable on angiography, aortography, or in an ECG-gated 64-slice cardiac CT, which corroborated the coronary course depicted by the angiogram.

SCA anomalies are uncommon (0.02–0.04%) and the variant we describe is exceedingly rare. To the best of our knowledge, this is the first reported case of this variant with evidence of a significant atherosclerotic lesion.

Panel A. The RCA arises normally in the right coronary sinus and continues to sequentially form the CX in the AV groove and continues on to form the LAD.

Panel B. A 90% lesion (L) is present in the mid-CX isolating the LAD.

Panel C. Aortography shows the RCA arising normally from the aorta (Ao), but no evidence of the left main coronary artery.

Panels D and E. Cardiac CT demonstrates the calcified RCA emanating from the right coronary sinus and seen later as the CX, which then continues anteriorly as the LAD (Panel F). CT reconstructions demonstrate continuity of the RCA and CX (Panel G), and that only the RCA arises from the aorta (Panel H).