Prevalence of heart failure and systolic ventricular dysfunction in older Australians: the Canberra Heart Study

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ABSTRACT

Objective: To estimate the prevalence of heart failure (HF) and left ventricular (LV) systolic dysfunction in a population-based sample of older Australians.

Design, setting and participants: A cross-sectional survey of 2000 randomly selected residents of Canberra, aged 60–86 years, conducted between February 2002 and June 2003. Participants were assessed by history, physical examination by a cardiologist, and echocardiography.

Main outcome measures: Age- and sex-specific prevalence rates of clinical HF and LV systolic dysfunction (defined as LV ejection fraction ⩽ 50%).

Results: Of 1846 people eligible for our study, 1388 (75%) agreed to participate and 1275 completed all investigations (mean age, 69.4 years; 50% men). In the study sample, 72 subjects (5.6%; 95% CI, 4.4%–7.1%) had clinical HF that had been previously diagnosed and was confirmed by our assessment. A further 0.6% (95% CI, 0.3%–1.2%) had undiagnosed clinical HF (ie, evidence of structural heart disease and symptoms/signs of cardiac insufficiency without a previous diagnosis of clinical HF). Thus, the overall prevalence of clinical HF in the sample was 6.3% (95% CI, 5.0%–7.7%). Clinical HF increased in prevalence with advancing age (a 4.4-fold increase from the 60–64-years age group to the 80–86-years age group; $P < 0.0001$). Of the 75 subjects (5.9%; 95% CI, 4.7%–7.3%) with LV systolic dysfunction, 44 (59%) were in the preclinical stage of disease.

Conclusion: Diagnosed HF cases represent the "tip of the iceberg" for the national burden of HF and LV systolic dysfunction. Clinically identifiable HF cases can remain undiagnosed, and the majority of people with LV systolic dysfunction are in a preclinical stage of the disease.

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The human and economic burden of heart failure (HF) in the community is expected to increase with the ageing of the Australian population. Even with the availability of effective medical therapy, the syndrome of HF is associated with substantial mortality, morbidity and economic cost (estimated to be over $1000 million in Australia in 2000).[1] Although HF, under the umbrella of "cardiovascular disease", has been nominated as a National Health Priority Area,[2] there are no population-based data with which to estimate the prevalence and incidence of HF and left ventricular (LV) dysfunction in Australia.[3] Such information is important to guide the allocation of health resources for managing HF and monitoring the impact of therapeutic and preventive strategies over time.

The aim of our study was to determine the prevalence of HF and LV systolic dysfunction in a population-based sample of older Australians.

METHODS

Study sample

Two thousand residents of southern Canberra, aged 60–85 years, were randomly selected from the January 2002 electoral roll, and invited by letter to participate in a cross-sectional echocardiographic survey. Institutionalised subjects and those who had died or had moved away from the Australian Capital Territory were excluded from the study sample ($n = 154$).

Participants were enrolled between February 2002 and June 2003. The sample size selected was based on precision of estimates to determine a prevalence of LV systolic dysfunction of 4% and the assumption of a 60% participation rate.

Echocardiographic assessment

Cardiac structure and function were assessed in all participants using transthoracic echocardiography (Acuson 128 XP/10, Siemens, Mountain View, Calif, USA) according to a standardised protocol.

Abnormal LV systolic function was categorised according to the LV ejection fraction (EF) (EF 41%–50%: mild LV systolic dysfunction; EF ⩽ 40%: moderate or severe LV systolic dysfunction).

Abnormal LV diastolic function was graded into three categories using Doppler evaluation of mitral and pulmonary venous inflow and tissue Doppler imaging of lateral mitral annulus motion (abnormal relaxation filling pattern: mild LV diastolic dysfunction; pseudonormal filling pattern: moderate LV diastolic dysfunction; restrictive filling pattern: severe LV diastolic dysfunction).[4]

LV mass was assessed by the area–length method[5] and indexed to height. Sex-specific reference limits (97.5th percentiles) were derived from the frequency distribution of indexed LV mass in a reference sample of subjects who had no history of ischaemic heart disease, hypertension, diabetes, atrial fibrillation or more than mild valvular heart disease and who were not taking cardioactive medications. Increased indexed LV mass was defined as > 132 g/m for women and > 157 g/m for men.

Mitral and aortic valvular function was assessed using Doppler and colour-Doppler echocardiography and graded according to

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1 Number (%) of participants with clinical heart failure and left ventricular (LV) systolic dysfunction, by age group and sex ($n = 1275$)

	Age group in years					
	60–64	65–69	70–74	75–79	80–86	Total
Clinical heart failure						
Men	7 (3.6)	12 (7.3)	6 (5.2)	16 (17.8)	11 (15.7)	52 (8.2)
Women	5 (2.6)	3 (2.0)	7 (4.8)	8 (7.8)	5 (10.4)	28 (4.4)
Total	12 (3.1)	15 (4.8)	13 (5.0)	24 (12.4)	16 (13.6)	80 (6.3)
LV systolic dysfunction						
Any dysfunction (EF ⩽ 50%)						
Men	11 (5.6)	13 (7.9)	10 (8.6)	9 (10)	12 (17.1)	55 (8.7)
Women	4 (2.1)	3 (2.0)	5 (3.4)	3 (2.9)	5 (10.4)	20 (3.1)
Total	15 (3.9)	16 (5.1)	15 (5.7)	12 (6.2)	17 (14.4)	75 (5.9)
Moderate or severe dysfunction (EF ⩽ 40%)						
Men	3 (1.5)	6 (3.7)	3 (2.6)	6 (6.7)	3 (4.3)	21 (3.3)
Women	2 (1.0)	0 (0.0)	1 (0.7)	1 (1.0)	2 (4.2)	6 (0.9)
Total	5 (1.3)	6 (1.9)	4 (1.5)	7 (3.6)	5 (4.2)	27 (2.1)

EF = ejection fraction. ◆

previously published criteria.[6] Valvular heart disease was defined as at least moderate stenosis or regurgitation of the mitral and/or aortic valves.

Structural heart disease was defined as LV systolic dysfunction, moderate or severe LV diastolic dysfunction, increased indexed LV mass and/or valvular heart disease.

Ascertaining HF clinical status

A self-reported history of clinical HF was verified by a review of the subject's medical records. During a consultation with a cardiologist who was blinded to the echocardiography findings and past medical history, participants were asked if they had symptoms of dyspnoea, orthopnea, paroxysmal nocturnal dyspnoea or dependant oedema. They were examined for the presence of increased heart rate, raised jugular venous pressure, displaced apex beat, added heart sounds, cardiac murmurs, lung crepitations or peripheral oedema. HF clinical status was ascertained according to the Framingham criteria for the clinical diagnosis of congestive heart failure.[7] Subjects with LV systolic dysfunction but no past history or clinical evidence of HF were considered to be in the preclinical phase of disease.

Evaluating clinical predictors of preclinical LV systolic dysfunction

A self-administered questionnaire was used to document a history of myocardial infarction, coronary disease, diabetes, hypertension, and alcohol consumption levels (none, low-risk, or high-risk [the latter defined as ⩾ 140 g/week for women and ⩾ 280 g/week for men[8]]).

Brachial artery systolic and diastolic blood pressure (Korotkoff phase V) were measured to the nearest 2 mmHg on the right arm using a standard mercury sphygmomanometer after 10 minutes of rest in a sedentary position. The measurement was repeated after 5 minutes and the two sets were averaged for each participant.

Hypertension was defined as systolic blood pressure ⩾ 140 mmHg, diastolic blood pressure ⩾ 90 mmHg, or the use of medical therapy for hypertension.

Diabetes was defined as a fasting serum glucose level ⩾ 7 mmol/L or the use of insulin or oral hypoglycaemic agents.

Height and weight were measured without shoes and in light clothing. Body mass index was calculated for each participant and categorised using the World Health Organization classification scheme (normal, < 25 kg/m^2; overweight, 25.0–29.9 kg/m^2; obese, ⩾ 30 kg/m^2).

Statistical analysis

Prevalence rates of LV systolic dysfunction and clinical HF were determined and stratified according to age and sex. The associations between clinical parameters and preclinical LV systolic dysfunction were examined in univariate analyses using χ^2 tests and in multivariable analyses using logistic regression to adjust for age and sex.

Ethics approval

Our study was approved by the human research ethics committees of ACT Health and Community Care and the Australian National University.

RESULTS

Of the 1846 people eligible for our study, 1388 (75%) agreed to participate. All participants provided written and informed consent. The only groups with participation rates under 70% were women aged 75–79 years (68%) and over 80 years (49%). Consequently, compared with the source population, the sample population had a higher proportion of men (50.5% v 47.2%;

2 Association of preclinical left ventricular systolic dysfunction with clinical characteristics

	Unadjusted OR (95% CI)	Adjusted OR (95% CI)*
Men	2.3 (1.2–4.4)	2.3 (1.2–4.4)†
Age group (referent: 60–69-years age group)		
70–79 years	1.2 (0.6–2.4)	1.3 (0.6–2.5)‡
80–86 years	3.2 (1.4–7.2)	3.0 (1.3–6.9)‡
Hypertension	1.2 (0.6–2.1)	1.1 (0.6–2.0)
Diabetes	0.3 (0.1–1.2)	0.3 (0.1–1.1)
Myocardial infarction	3.9 (1.6–9.7)	3.0 (1.2–7.5)
Coronary disease	2.1 (1.03–4.2)	1.6 (0.8–3.3)
Body mass index (referent: < 25 kg/m^2)		
25–29.9 kg/m^2	0.6 (0.3–1.2)	0.5 (0.3–1.1)
⩾ 30 kg/m^2	0.7 (0.3–1.5)	0.8 (0.3–1.6)
High-risk alcohol intake	1.0 (0.2–4.4)	1.0 (0.2–4.4)

OR = odds ratio. * Adjusted for age and sex unless otherwise specified; † Adjusted for age. ‡ Adjusted for sex. ◆

$P < 0.012$) and were younger (68.9 years v 69.6 years; $P = 0.0005$). Ninety-two per cent of participants (1275/1388; mean age, 69.4 years; 50% men; 97.6% white) completed all the investigations relevant to our study. Of these, 269 participants (21.1% [95% CI, 18.8%–24.4%]) had evidence of structural heart disease; 75 (5.9% [95% CI, 4.7%–7.3%]) had LV systolic dysfunction; 72 (5.6%) had moderate or severe diastolic dysfunction with EF > 50%; 102 (8.0%) had increased LV mass without impaired systolic function or moderate/severe diastolic function; and 20 (1.6%) had aortic and/or mitral valvular heart disease.

Age- and sex-specific prevalence rates of clinical HF and LV systolic dysfunction are presented in Box 1. Of those with any LV systolic dysfunction, 44 (59%) were at a preclinical stage. However, only six of the 27 subjects (22%) with moderate or severe LV systolic dysfunction were in the preclinical phase of disease. Increasing age, male gender, and a history of myocardial infarction were associated with preclinical LV systolic dysfunction (Box 2).

Although diabetes was associated with clinical systolic dysfunction (odds ratio [OR], 2.6; 95% CI, 1.2–5.7), there was a trend toward a lower likelihood of preclinical LV systolic dysfunction in people with diabetes (OR, 0.26; 95% CI, 0.1–1.1). There were no significant associations between preclinical LV systolic dysfunction and hypertension, body mass index category or alcohol intake status.

In the study sample, 72 subjects (5.6%; 95% CI, 4.4%–7.1%) had clinical HF that had been previously diagnosed and was validated by our assessment. Of these, approximately 60% had preserved LV systolic function (EF > 50%), a finding that was more frequent in women than men (77% v 52%; $P = 0.039$).

A further eight participants (0.6%; 95% CI, 0.3%–1.2%) without a previous diagnosis of clinical HF had evidence of structural heart disease and symptoms/signs of cardiac insufficiency (ie, undiagnosed clinical HF). Thus, the overall prevalence of clinical HF in the sample was 6.3% (95% CI, 5.0%–7.7%). Clinical HF was more common in men than women (8.2% v 4.4%; $P = 0.005$) and increased in prevalence with advancing age, with a 4.4-fold increase from the 60–64-years age group to the 80–86-years age group ($P < 0.0001$). The proportion of undiagnosed clinical HF cases was not related to age ($P = 0.34$) or sex ($P = 0.54$).

DISCUSSION

Our study provides the first population-based estimates of HF prevalence in Australia. Prior estimates of HF prevalence and incidence have been derived by extrapolation from overseas data,[1] hospital separations, or studies on clinic-based samples in which a minority of patients had echocardiographic confirmation of underlying structural heart disease.[9]

Our results suggest that diagnosed cases of HF represent the "tip of the iceberg" for the national burden of HF and structural heart disease. Clinically identifiable HF cases can remain undiagnosed, and although 21.1% of our sample had structural heart disease, only 6.3% had clinical HF. Moreover, of the 5.9% of participants with LV systolic dysfunction, 59% were in the preclinical stage of disease.

Comparison of our findings with international studies is limited by differences in study methods and outcome definitions. Framingham investigators have reported that about 6%–10% of the population aged over 65 years experience clinical HF.[10] However, in early epidemiological cardiovascular surveys such as the Framingham study, HF status was ascertained using clinical scores that had relatively poor sensitivity and specificity for structural heart disease. It has only been during the past decade that surveys have incorporated echocardiography to confirm structural heart disease in people with non-specific symptoms and signs of cardiac insufficiency.

The burden of LV systolic dysfunction in our sample is consistent with estimates from the Olmsted County,[11] Rotterdam[12] and

3 Comparison of results from prevalence surveys of left ventricular (LV) systolic dysfunction

Study	Sample size (% men)	Mean age in years (range)	Participation rate	Identification of congestive heart failure	Method of measuring EF	Definition of abnormal LV systolic function	Prevalence (%)
Glasgow[14]	1467 (48)	50 (25–75)	56%*	MRC dyspnoea class; use of diuretics	2D	EF < 35%	7.7
Rotterdam[12]	2267 (45)	66 (55–94)	78%	Two-step approach based on (i) WHO dyspnoea class, clinical examination; (ii) indication for cardiac medications	M-mode	EF < 42.5%	3.7
Olmsted County[11]	2042 (48)	63 (45–96)	47%	Medical records	M-mode, 2D, and visual estimate	EF ⩽ 50%	6.0
						EF ⩽ 40%	2.0
Strong Heart[15]	3184 (37)	58 (49–78)	62%	Unspecified	M-mode	EF ⩽ 54%	13.9
						EF < 40%	2.9
ECHOES[13]	3960 (50)	61 (⩾ 45)	63%	New York Heart Association classification	2D	EF ⩽ 50%	5.3
						EF < 40%	1.8
Canberra†	1275 (50)	69 (60–86)	75%	Medical records and clinical examination	2D	EF ⩽ 50%	5.9
						EF ⩽ 40%	2.1

EF = ejection fraction. MRC = Medical Research Council (UK). WHO = World Health Organization. * Based on 83% response rate to invitation to participate in parent survey and 67% response rate to echocardiographic study. † The study reported here. ◆

"ECHOES"[13] surveys, conducted in predominantly white populations with similar cardiovascular risk profiles (Box 3). However, the prevalence rates of LV systolic dysfunction in our study were lower than the estimates from the Strong Heart study,[15] conducted exclusively in a population of Native Americans, and the Glasgow study,[14] whose population has a high prevalence of cardiovascular disease. Like the surveys from the United States and Northern Europe,[11-15] our results show that most cases of LV systolic dysfunction in the community are preclinical.

Strengths of our study are its population-based sample and good participation rate. However, a number of factors may limit the generalisability of our results. Firstly, women aged 75 years and over were under-represented in our sample. This may have resulted in an underestimation of disease prevalence in the source population. Secondly, almost all study participants were white, so our results may not be applicable to non-white populations. Thirdly, the National Health Survey has documented that ACT residents have lower rates of classical risk factors for cardiovascular disease than the national average,[16] which may reduce the prevalence of HF in this population compared with national rates. On the other hand, ready access to medical therapy for acute coronary syndromes and hypertension in this urban setting may paradoxically increase the prevalence of structural heart disease and HF, through a survivor effect.

Our results have several important public health implications. Firstly, with the availability of effective medical therapy for patients with clinical HF, it is crucial to optimise methods of diagnosing the condition in the community. Using strict criteria for the clinical diagnosis of congestive HF, we found that 10% of people with structural heart disease and clinical evidence of HF had not been previously diagnosed with the condition. The National Institute of Clinical Studies has recently evaluated barriers to the correct diagnosis of HF and the implementation of proven management strategies in primary care.[17] Solutions based on targeted education of health professionals and patients, and the increased use of diagnostic tools such as echocardiography, have been proposed.

Secondly, as secondary preventive measures have been shown to be effective for patients with preclinical LV systolic dysfunction, methods for identifying affected patients should also be optimised. Targeted screening programs of high-risk people (such as men, older age groups, and patients with documented coronary disease or prior myocardial infarction) should be evaluated for their cost-effectiveness.

Thirdly, with the anticipated increase in the proportion of the population aged over 60 years (among whom most HF cases arise) and escalating rates of obesity and diabetes in the community, there seems little hope of stemming the rise in HF cases to epidemic proportions unless primary preventive efforts directed at the individual and population level are adopted.

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COMPETING INTERESTS

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