The effect of evidence-based medication use on long-term survival in patients hospitalised for heart failure in Western Australia

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ABSTRACT

Objectives: To examine trends and predictors of prescription medications on discharge after first (index) hospitalisation for heart failure (HF), and the effect on all-cause mortality of evidence-based therapy.

Design: A retrospective multicentre cohort study, with medical record review.

Setting: Three tertiary-care hospitals in Perth, Western Australia.

Patients: WA Hospital Morbidity Data were used to identify a random sample of 1006 patients with an index admission to hospital for HF between 1996 and 2006.

Main outcome measures: Proportion of patients prescribed evidence-based therapy for HF on discharge from hospital; and 1-year all-cause mortality.

Results: Among 944 patients surviving to hospital discharge, the prescription rate of angiotensin-converting enzyme (ACE) inhibitors/angiotensin receptor blockers (ARBs) (74.3%) and loop diuretics (85.5%) remained high over the study period, whereas that of β-blockers and spironolactone increased (10.5% to 51.3% and 1.4% to 23.3%, respectively), and digoxin prescription decreased (38.1% to 20.7%). The temporal trends in use of β-blockers, spironolactone and digoxin were in line with clinical trial evidence. Age ⩾ 75 years was a significant, negative predictor of β-blocker and spironolactone prescription. In-hospital echocardiography, performed in 53% of patients, was associated with a significantly greater likelihood of treatment with ACE inhibitors/ARBs, β-blockers and spironolactone. Both ACE inhibitors/ARBs and β-blockers prescribed on discharge were associated with a lower adjusted hazard ratio (HR) for mortality at 1-year (HR, 0.71; $P = 0.003$; and HR, 0.68; $P = 0.002$, respectively).

Conclusion: ACE inhibitors/ARBs and β-blockers, prescribed during initial hospitalisation for HF, are associated with improved long-term survival. Therapy became more evidence based over the study period, but echocardiography, an important predictor of evidence-based therapy, was underutilised.

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In Australia, as in other countries, heart failure (HF) is one of the most common and important causes of hospitalisation and death.[1-3] However, the results of large randomised controlled trials conducted in the past two decades have brought about considerable progress in HF treatment.[1,3] Inhibition of the neurohormonal pathways with angiotensin-converting enzyme (ACE) inhibitors and β-blockers has been shown to decrease the risk of death and rehospitalisation;[4,5] angiotensin receptor blockers (ARBs) and aldosterone antagonists improve survival in selected patients with systolic HF;[6,7] and digoxin, although not affecting survival in patients with HF and sinus rhythm, is associated with a reduction in hospitalisation.[8]

Studies in Australia and other countries have documented suboptimal use of evidence-based therapy in a broad range of patients with HF.[9-13] This may be because many patients who develop HF are elderly, often female, have preserved left ventricular (LV) systolic function, and are not representative of the patients with HF included in clinical trials.[9,14] Another contributory factor is failure to initiate evidence-based therapy in hospital, as physicians may be reluctant to start therapy not initiated in hospital.[15]

We aimed (i) to ascertain whether there was optimal use of evidence-based therapy during a first (index) admission for HF to a tertiary-care hospital; (ii) to determine the predictors of evidence-based therapy at discharge; and (iii) to assess the independent effect on long-term survival of treatment at discharge with ACE inhibitors/ARBs, β-blockers, and aldosterone antagonists.

METHODS

Sample selection

The Western Australia Hospital Morbidity Data (WA HMD)[16] were used to identify a random sample of 1006 patients with an index admission for HF to one of the three tertiary-care hospitals in Perth, WA, between 1996 and 2006. An index admission for HF was defined as a principal diagnosis of HF (WA HMD), and no prior admission for HF in the previous 5 years. The coding for HF in the WA HMD was validated in the same random sample: a principal diagnosis of HF had a positive predictive value of 92.4% when compared with the Boston criteria for "definite" HF.[17]

Data collection

The medical charts were reviewed by two trained researchers, who extracted data on demographic characteristics, hospital discharge diagnosis codes, vital signs on admission, HF score (Boston criteria),[17] results of an in-hospital echocardiogram, blood test results, and medications prescribed on discharge. Comorbid conditions, specifically ischaemic heart disease, hypertension, diabetes, atrial fibrillation, chronic airway limiting disease, and renal failure were also abstracted from the records.

Definitions

In the subgroup of patients ($n = 537$) who had an in-hospital echocardiogram, preserved systolic function was defined as an LV ejection fraction > 40%, or LV systolic function described as normal or mildly impaired. LV systolic dysfunction was defined as an LV ejection fraction < 40%, or moderate or severe impairment of LV systolic function (Box 1).

Anaemia was defined as a haemoglobin level on admission of < 13.0 g/dL in men and < 12.0 g/dL in women (World Health Organization [WHO] criteria).[18] Kidney disease was defined as an estimated glomerular filtration rate (eGFR) of < 60 mL/min/1.73 m^2, using the Modification of Diet in Renal Disease equation.[19]

Primary end points

The main outcome measures were the proportion of patients prescribed specific medications for HF on discharge from hospital, and the all-cause mortality. The WA HMD were linked to the death register in WA to

1 Baseline characteristics and sex differences in the study cohort

Description	Total	Men	Women	*P*
Number of patients	1006	508 (50.5%)	498 (49.5%)	ns
Mean age (SD), years	76.3 (12.8)	73.0 (12.8)	77.5 (12.4)	< 0.001
Age ⩾ 75 years	654 (65.0%)	295 (58.0%)	359 (72.1%)	< 0.001
Medical history				
Hypertension	581 (57.8%)	275 (54.1%)	306 (61.4%)	0.054
Ischaemic heart disease	528 (52.5%)	288 (56.7%)	239 (48.0%)	0.005
Atrial fibrillation	312 (31.0%)	154 (30.3%)	158 (31.7%)	ns
Valvular heart disease	120 (11.9%)	47 (9.3%)	73 (14.7%)	0.019
Diabetes mellitus	292 (29.0%)	172 (33.9%)	120 (24.1%)	0.002
Renal failure (clinical diagnosis)	196 (19.5%)	121 (23.8%)	75 (15.1%)	0.001
Chronic airway limiting disease	190 (18.9%)	116 (22.8%)	74 (14.9%)	0.005
Clinical signs on admission				
Systolic BP > 140 mmHg	454 (45.1%)	200 (39.4%)	254 (51.0%)	< 0.001
Mean (SD) systolic BP, mmHg	143.6 (31.4)	140.0 (30.7)	147.3 (31.7)	< 0.001
Mean (SD) heart rate	93.6 (24.3)	91.4 (23.9)	95.7 (24.6)	0.005
Mean (SD) Boston score[17]	10.4 (2.0)	10.3 (2.0)	10.5 (1.9)	ns
Laboratory values				
Mean (SD) haemoglobin level, g/dL	12.6 (2.1)	12.9 (2.2)	12.4 (1.9)	< 0.001
Anaemia (WHO criteria)[18]	459 (45.6%)	255 (50.2%)	204 (41.0%)	0.004
Mean (SD) eGFR mL/min/1.73 m^2	57.3 (26.6)	58.6 (27.2)	55.9 (25.8)	ns
eGFR < 60 mL/min/1.73 m^2	593 (58.9%)	295 (58.1%)	298 (59.8%)	ns
Echocardiography				
Number of patients	537 (53.4%)	293 (57.7%)	244 (49.0%)	0.006
LV ejection fraction, % (SD) (*n* = 203)	42.8% (19.8%)	38.9% (18.3%)	47.8% (24.3%)	0.002
LV function				
Normal	194 (36.1%)	73 (24.9%)	121 (49.6%)	< 0.001
Mild dysfunction	103 (19.2%)	63 (21.5%)	40 (16.4%)	
Moderate dysfunction	127 (23.6%)	79 (27.0%)	48 (19.7%)	
Severe dysfunction	113 (21.0%)	78 (26.6%)	35 (14.3%)	
Medications at discharge in those surviving to discharge (*n* = 944 [476 men, 468 women])				
ACE inhibitors/ARBs	701 (74.3%)	358 (75.2%)	343 (73.3%)	ns
β-Blockers	318 (33.7%)	166 (34.9%)	152 (32.5%)	ns
Spironolactone	154 (16.3%)	87 (18.3%)	67 (14.3%)	ns
Digoxin	288 (30.5%)	139 (29.2%)	149 (31.8%)	ns
Loop diuretics	807 (85.5%)	405 (85.1%)	402 (85.9%)	ns
Statins	256 (27.1%)	149 (31.3%)	107 (22.9%)	0.004
Aspirin	496 (52.5%)	262 (55.0%)	234 (50.0%)	ns
Cumulative mortality				
1-year mortality	282 (28.0%)	149 (29.3%)	133 (26.7%)	ns
3-year mortality	446 (44.3%)	224 (44.1%)	222 (44.6%)	ns

Continuous variables are expressed as mean (SD); categorical variables are expressed as proportions (number [%]).
ns = not significant. BP = blood pressure. WHO = World Health Organization. LV = left ventricular.
eGFR = estimated glomerular filtration rate. ACE = angiotensin converting enzyme.
ARBs = angiotensin receptor blockers. ◆

ascertain deaths in the cohort to the end of the follow-up period (31 December 2006).

Ethics approval

Ethics approval was obtained from the human research ethics committees of the three Perth tertiary-care hospitals, the University of WA, and the WA Department of Health.

Statistical analysis

For descriptive analyses and comparison of trends for the overall cohort, the study period was divided into four: 1996–1998 (baseline), 1999–2001, 2002–2004 and 2005–2006. Multivariable logistic regression models were used to determine predictors of treatment at discharge with ACE inhibitors/ARBs, β-blockers or spironolactone. The association between medications prescribed and death at 1 year and 3 years was tested using the Cox proportional hazards model, adjusting for baseline characteristics and other covariates (Box 1). The odds ratio or hazard ratio and 95% confidence intervals are reported. For the 1-year survival analysis, we included all cohort patients surviving to discharge; the 3-year survival analysis included all those with a 3-year follow-up. Statistical analyses were performed with Stata, version 10 (StataCorp, College Station, Tex, USA).

RESULTS

The study cohort consisted of 1006 patients with a mean age (SD) of 76.3 (12.8) years; 49.5% were women. Baseline characteristics are compared in Box 1. The women were older than the men; hypertension and ischaemic heart disease were the most common comorbidities, with women more likely to have hypertension and men more likely to have ischaemic heart disease (Box 1).

Echocardiography

Overall, 53.4% of patients had an in-hospital echocardiogram; there was no increase in use of echocardiography over the study period. Older age and female sex were negative predictors of an in-hospital echocardiogram. Preserved systolic function (55.3% of the cohort) was more common in women than men (66.0% v 46.4%, $P < 0.001$) (Box 1). The cumulative 1-year mortality was higher in patients who did not have an echocardiogram compared with those who did (35.4% v 21.6%; $P < 0.001$).

Medications prescribed on discharge

Of the 944 patients surviving to hospital discharge, prescription rates of medications on discharge are shown in Box 1 and Box 2. Over the study period, the high prescription rate of ACE inhibitors/ARBs (74.3%) and loop diuretics (85.5%) did not change, but prescription rates increased for β-blockers (10.5% to 51.3%), and spironolactone (1.4% to 23.3%), and decreased for digoxin (38.1% to 20.7%). The same prescription trends were seen in the subgroups of patients with LV systolic dysfunction or preserved systolic function (Box 2). How-

2 Trends in prescription of medications at discharge in patients with heart failure who survived to hospital discharge, 1996–2006

Therapeutic groups	Total	1996–1998	1999–2001	2002–2004	2005–2006	*P* (for trend)
All patients (*n* = 944)						
ACE inhibitors/ARBs	74.3%	73.8%	75.2%	75.3%	72.0%	ns
β–Blockers	33.7%	10.5%	28.4%	43.6%	51.3%	< 0.001
Spironolactone	16.3%	1.4%	13.0%	25.4%	23.3%	< 0.001
Digoxin	30.5%	38.1%	30.3%	31.7%	20.7%	0.002
Loop diuretics	85.5%	89.5%	86.6%	82.9%	83.4%	ns
Patients with LV systolic dysfunction on echocardiography (*n* = 225)						
ACE inhibitors/ARBs	86.7%	89.3%	80.7%	89.4%	87.8%	ns
β–Blockers	44.4%	21.4%	35.5%	54.6%	73.2%	< 0.001
Spironolactone	28.4%	0.0	22.6%	47.0%	46.3%	< 0.001
Digoxin	36.4%	51.8%	37.1%	34.9%	17.1%	0.006
Loop diuretics	91.1%	92.9%	91.9%	87.9%	92.7%	ns
Patients with preserved systolic function on echocardiography (*n* = 284)						
ACE inhibitors/ARBs	74.3%	71.7%	74.3%	73.5%	77.3%	ns
β–Blockers	35.6%	2.2%	36.5%	43.9%	45.5%	< 0.001
Spironolactone	14.8%	0.0	14.9%	19.4%	18.2%	0.017
Digoxin	28.9%	43.5%	21.6%	31.6%	22.7%	0.041
Loop diuretics	85.2%	91.3%	82.4%	83.7%	86.4%	ns
Patients without assessment of LV function by echocardiography (*n* = 435)						
ACE inhibitors/ARBs	67.8%	66.7%	72.9%	69.1%	60.5%	ns
β-Blockers	26.9%	8.3%	19.5%	37.4%	45.4%	< 0.001
Spironolactone	11.0%	2.8%	6.8%	18.7%	16.3%	< 0.001
Digoxin	28.5%	28.7%	32.2%	30.1%	20.9%	ns
Loop diuretics	82.8%	87.0%	86.4%	79.7%	76.7%	ns

ACE = angiotensin converting enzyme; ARBs = angiotensin receptor blockers; LV = left ventricular; ns = not significant. ◆

ever, patients with LV systolic dysfunction, compared with those with preserved systolic function, were more likely to be prescribed ACE inhibitors/ARBs, β-blockers, spironolactone, and digoxin (Box 2).

Independent predictors of evidence-based therapy

The odds of being prescribed β-blockers and spironolactone increased over the successive time periods from 1999 onwards (Box 3). In patients having LV function assessed by echocardiography, there was a higher rate of prescription of ACE inhibitors/ARBs, β-blockers and spironolactone (averaging 73%, 43%, and 74%, respectively) (Box 3). Negative predictors of evidence-based therapy were age group ⩾ 75 years (β-blockers and spironolactone), renal failure (ACE inhibitors/ARBs and spironolactone), and chronic airway limiting disease (β-blockers). Sex was not a significant predictor of evidence-based therapy.

Discharge medication and 1-year and 3-year survival

Characteristics independently predictive of mortality are listed in Box 4. Patients prescribed ACE inhibitors/ARBs and β-blockers on discharge had a significantly lower (30% or lower) 1- and 3-year adjusted hazard of death (Box 4). Prescription of spironolactone and digoxin was not associated with survival.

In an analysis restricted to patients having echocardiography in hospital, both ACE inhibitors/ARBs (hazard ratio [HR], 0.45; 95% CI, 0.26–0.78; $P = 0.004$) and β-blockers (HR, 0.53; 95% CI, 0.32–0.87; $P = 0.011$) were associated with a reduction in the adjusted mortality hazard at 1-year in patients with LV systolic dysfunction. In patients with preserved systolic function, only β-blockers (HR, 0.62; 95% CI, 0.39–0.99; $P = 0.048$) were associated with a lower adjusted mortality hazard at 1-year.

DISCUSSION

The prescription rates of β-blockers and aldosterone antagonists on hospital discharge, increased significantly from 1996 to 2006 in our cohort of patients with HF, while prescription rates of ACE inhibitors/ARBs were consistently high. These trends reflect clinical trial evidence. Performance of an echocardiogram during index hospitalisation for HF was a key predictor of evidence-based therapy. Yet, in just under half of our cohort, echocardiography was not performed in hospital and this rate did not change over the study period. Prescription of ACE inhibitors/ARBs and β-blockers on discharge were both independently associated with better long-term survival. These results are of clinical importance, given the potential impact of proven pharmacotherapies on the morbidity and mortality of HF.

Studies in Australia, as elsewhere, have found suboptimal use of evidence-based therapy in patients with HF.[9-13] A contributing factor to the evidence–treatment gap is failure to initiate this therapy during hospitalisation.[20] A recent Danish hospital registry study showed that early and continuing treatment with proven pharmacotherapies for HF produced long-term survival benefits.[21]

Our cohort's prescription rate of ACE inhibitors/ARBs on discharge (74.3%) was better than the 58.7% rate in the Australian CHART study (1998–2000).[13] However, other contemporary studies (the ADHERE registry[9] and the EuroHeart Failure Survey II[14]) have reported higher ACE inhibitor/ARB discharge prescription rates (83% and 80.2%, respectively) for hospitalised patients with HF. In the subgroup of patients with LV systolic dysfunction in our cohort, the proportion taking ACE inhibitors/ARBs reached a comparable 86.7%, despite renal insufficiency (causing intolerance to these drugs) being common among patients with HF. We found that the prescription of ACE inhibitors/ARBs at discharge was an independent predictor of improved long-term survival in our entire cohort, with the exception of the subgroup of patients with preserved systolic function, as also found in clinical trials.[22]

Despite the nearly fivefold increase in use of β-blockers over the study period, in line with recent clinical trial evidence,[5] β-blockers may have been underutilised in our cohort. The 51.3% rate of β-blocker use by 2005–2006 was lower than that in other studies (ADHERE, 80.1% in patients with LV systolic dysfunction;[9] and EuroHeart

3 Predictors of discharge therapy with ACE inhibitors/ARBs, β-blockers and spironolactone

Independent variable	Odds ratio (95% CI)	*P*
Prescription of ACE inhibitors/ARBs		
Boston score[17]	1.15 (1.06–1.23)	< 0.001
Renal failure	0.68 (0.47–0.99)	0.045
Anaemia (WHO criteria)[18]	0.68 (0.50–0.93)	0.017
LV assessment by echocardiography	1.73 (1.27–2.36)	0.001
Prescription of β-blockers		
Age group < 65 years	1.00	
Age group 65–74 years	0.73 (0.45–1.20)	0.22
Age group ⩾ 75 years	0.50 (0.32–0.76)	0.001
Period 1996–1998	1.00	
Period 1999–2001	3.30 (1.92–5.66)	< 0.001
Period 2002–2004	7.14 (4.18–12.20)	< 0.001
Period 2005–2006	10.75 (6.13–18.86)	< 0.001
Ischaemic heart disease	2.07 (1.52–2.83)	< 0.001
Chronic airway limiting disease	0.30 (0.19–0.47)	< 0.001
Prescription of ACE inhibitors/ARBs	1.81 (1.26–2.62)	0.001
Prescription of spironolactone	1.57 (1.05–2.35)	0.027
LV assessment by echocardiography	1.43 (1.05–1.96)	0.025
Prescription of spironolactone		
Age group < 65 years	1.00	
Age group 65–74 years	0.56 (0.31–0.99)	0.046
Age group ⩾ 75 years	0.46 (0.28–0.74)	0.001
Period 1996–1998	1.00	
Period 1999–2001	9.69 (2.90–32.39)	< 0.001
Period 2002–2004	23.03 (7.0–75.5)	< 0.001
Period 2005–2006	19.4 (5.78–64.77)	< 0.001
Renal failure	0.55 (0.33–0.92)	0.022
Prescription of β-blockers	1.48 (1.00–2.17)	0.047
LV assessment by echocardiography	1.74 (1.17–2.58)	0.006

ACE = angiotensin converting enzyme. ARBs = angiotensin receptor blockers. WHO = World Health Organization. LV = left ventricular. ◆

4 Adjusted 1-year and 3-year mortality in patients surviving to hospital discharge, using multivariable Cox proportional hazards models

Variable	Hazard ratio (95% CI)	*P*
1-year survival (*n* = 932)		
Age	1.04 (1.03–1.05)	< 0.001
Female sex	0.82 (0.67–1.01)	0.067
Presence of anaemia (WHO criteria)[18]	1.49 (1.22–1.83)	< 0.001
Presence of kidney disease	1.57 (1.25–1.96)	< 0.001
Systolic blood pressure	0.99 (0.99–1.00)	0.014
Ischaemic heart disease	1.23 (1.00–1.51)	0.053
Discharge prescription		
ACE inhibitors/ARBs	0.71 (0.57–0.89)	0.003
β-Blockers	0.68 (0.53–0.86)	0.002
Spironolactone	0.87 (0.64–1.20)	0.390
3-year survival (*n* = 651)		
Age	1.05 (1.04–1.07)	< 0.001
Female sex	0.73 (0.57–0.93)	0.010
Presence of anaemia (WHO criteria)[18]	1.57 (1.25–1.98)	< 0.001
Presence of kidney disease	1.53 (1.18–1.99)	0.001
Ischaemic heart disease	1.30 (1.03–1.64)	0.030
Number of comorbidities	1.11 (1.00–1.22)	0.040
Systolic blood pressure	0.99 (0.99–1.14)	0.003
Boston score[17]	1.06 (1.00–1.14)	0.062
Discharge prescription		
ACE inhibitors/ARBs	0.65 (0.50–0.83)	0.001
β-Blockers	0.71 (0.53–0.95)	0.020
Spironolactone	0.88 (0.59–1.31)	0.540

ACE = angiotensin converting enzyme. ARBs = angiotensin receptor blockers. WHO = World Health Organization. ◆

Failure Survey II, 61.4%[14]), but higher than in the CHART study (24.0%).[13]

Some of the prescription rate differences between studies may relate to inclusion of patients with preserved systolic function, in whom β-blockers have not been clearly shown to influence mortality or morbidity.[22,23] This may also explain the higher prescription rate of β-blockers in patients with LV systolic dysfunction, versus those with preserved systolic function, in our cohort. The overall 50% lower likelihood of β-blocker prescription in patients aged ⩾ 75 years (Box 3) also suggests underutilisation, given this drug's demonstrated benefit and tolerability, even in elderly patients with HF.[23] Clinicians need to ensure that all patients with systolic HF, regardless of age, receive β-blockers, unless the drug is specifically contraindicated. Even patients with chronic airway limiting disease tolerate and benefit from β-blockers to the same extent as those without this condition.[24] Crucially, prescription of β-blockers at discharge was independently associated with survival in our whole cohort, as well as in the subgroups of patients with LV systolic dysfunction or preserved systolic function. The survival benefit in the latter subgroup may be explained by inclusion of patients with mild LV systolic impairment within the definition of preserved systolic function.

The 1999 Randomized Aldactone Evaluation Study (RALES) showed a survival benefit from adding spironolactone to ACE inhibitors and loop diuretics in patients with severe heart failure and an LV ejection fraction < 35%.[7] Accordingly, the rate of discharge prescription of spironolactone in our cohort increased from 1.4% to 23.3% (46.3% in the subgroup with LV systolic dysfunction) after the RALES was published. Our results did not show an independent association of spironolactone with 3-year mortality (Box 4), but this was not unexpected due to the restricted number of patients prescribed aldosterone antagonists (recommended for patients who remain severely symptomatic, despite appropriate doses of ACE inhibitors and diuretics).[1,3] Older age, independent of renal failure, was a negative predictor of

spironolactone prescription, although age was not a specific contraindication for the RALES.[7] However, caution is required, as spironolactone use has been associated with an increased risk of hyperkalaemia and adverse outcomes in patients with chronic HF.[25]

Prescribing of digoxin at discharge declined substantially over the study period, with the 1997 Digitalis Investigation Group trial[8] showing that digoxin should be a second-line agent after ACE inhibitors and β-blockers in patients with HF and sinus rhythm.[1,3] As expected, we found no significant association between digoxin prescription and 3-year mortality, but its effect on rehospitalisation was not assessed.

The clinical similarities between the two main forms of HF (preserved systolic function and LV systolic dysfunction), and their differential evidence base for treatment, have been reported.[22] Echocardiography is the most valuable tool for differentiating between these two syndromes, as well as determining LV dysfunction severity,[22] as emphasised by the guidelines for diagnosis and management of suspected HF.[1,3] Yet, only half of our patients (all of whom had been admitted with HF for the first time) had an in-hospital echocardiogram, and its use did not increase over the study period. We confirmed the independent association between echocardiography and a higher prescription rate of ACE inhibitors/ARBs, β-blockers and aldosterone antagonists, but echocardiography was more often performed in younger patients, rather than older patients most in need of optimal HF treatment.

Our study's limitations are its observational nature, involving retrospective medical chart reviews. The substudy of patients with an in-hospital echocardiogram was limited by a small sample size. Long-term compliance with discharge medications and whether drug doses were optimal was not assessed. Our findings cannot be generalised to non-tertiary-care hospitals, where uptake and adherence to evidence-based guidelines may be inferior. Our study's strengths are that the principal diagnosis of HF was validated against the Boston diagnostic criteria;[17] the comprehensive clinical and biochemical data allowed adjustment for confounders of mortality; and, importantly, our study observed clinical practice during the contemporary era of HF management.

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COMPETING INTERESTS

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REFERENCES

1 Krum H, Jelinek MV, Stewart S, et al. Guidelines for the prevention, detection and management of people with chronic heart failure in Australia 2006. *Med J Aust* 2006; 185: 549-557.
2 Clark RA, McLennan S, Dawson A, et al. Uncovering a hidden epidemic: a study of the current burden of heart failure in Australia. *Heart Lung Circ* 2004; 13: 266-273.
3 Hunt SA, Baker DW, Chin MH, et al. ACC/AHA guidelines for the evaluation and management of chronic heart failure in the adult: executive summary. *Circulation* 2001; 104: 2996-3007.
4 Garg R, Yusuf S. Overview of randomized trials of angiotensin-converting enzyme inhibitors on mortality and morbidity in patients with heart failure. Collaborative Group on ACE Inhibitor Trials. *JAMA* 1995; 273: 1450-1456.
5 Brophy JM, Joseph L, Rouleau JL. Beta-blockers in congestive heart failure. A Bayesian meta-analysis. *Ann Intern Med* 2001; 134: 550-560.
6 Cohn JN, Tognoni G. A randomized trial of the angiotensin-receptor blocker valsartan in chronic heart failure. *N Engl J Med* 2001; 345: 1667-1675.
7 Pitt B, Zannad F, Remme WJ, et al. The effect of spironolactone on morbidity and mortality in patients with severe heart failure. Randomized Aldactone Evaluation Study Investigators. *N Engl J Med* 1999; 341: 709-717.
8 The effect of digoxin on mortality and morbidity in patients with heart failure. The Digitalis Investigation Group. *N Engl J Med* 1997; 336: 525-533.
9 Fonarow GC, Heywood JT, Heidenreich PA, et al. Temporal trends in clinical characteristics, treatments, and outcomes for heart failure hospitalizations, 2002 to 2004: findings from Acute Decompensated Heart Failure National Registry (ADHERE). *Am Heart J* 2007; 153: 1021-1028.
10 Cleland JG, Cohen-Solal A, Aguilar JC, et al. Management of heart failure in primary care (the IMPROVEMENT of Heart Failure Programme): an international survey. *Lancet* 2002; 360: 1631-1639.
11 Krum H, Tonkin AM, Currie R, et al. Chronic heart failure in Australian general practice. The Cardiac Awareness Survey and Evaluation (CASE) Study. *Med J Aust* 2001; 174: 439-444.
12 Lowe JM, Candlish PM, Henry DA, et al. Management and outcomes of congestive heart failure: a prospective study of hospitalised patients. *Med J Aust* 1998; 168: 115-118.
13 Wlodarczyk JH, Keogh A, Smith K, McCosker C. CHART: congestive cardiac failure in hospitals, an Australian review of treatment. *Heart Lung Circ* 2003; 12: 94-102.
14 Nieminen MS, Brutsaert D, Dickstein K, et al. EuroHeart Failure Survey II (EHFS II): a survey on hospitalized acute heart failure patients: description of population. *Eur Heart J* 2006; 27: 2725-2736.
15 Fonarow GC, Gheorghiade M, Abraham WT. Importance of in-hospital initiation of evidence-based medical therapies for heart failure — a review. *Am J Cardiol* 2004; 94: 1155-1160.
16 Holman CD, Bass AJ, Rouse IL, Hobbs MS. Population-based linkage of health records in Western Australia: development of a health services research linked database. *Aust N Z J Public Health* 1999; 23: 453-459.
17 Teng T-H, Finn J, Hung J, et al. A validation study: how effective is the hospital morbidity data as a surveillance tool for heart failure in Western Australia? *Aust N Z J Public Health* 2008; 32: 405-407.
18 Izaks GJ, Westendorp RG, Knook DL. The definition of anemia in older persons. *JAMA* 1999; 281: 1714-1717.
19 Levey AS, Bosch JP, Lewis JB, et al. A more accurate method to estimate glomerular filtration rate from serum creatinine: a new prediction equation. Modification of Diet in Renal Disease Study Group. *Ann Intern Med* 1999; 130: 461-470.
20 Whellan DJ, Gaulden L, Gattis WA, et al. The benefit of implementing a heart failure disease management program. *Arch Intern Med* 2001; 161: 2223-2228.
21 Gislason GH, Rasmussen JN, Abildstrom SZ, et al. Persistent use of evidence-based pharmacotherapy in heart failure is associated with improved outcomes. *Circulation* 2007; 116: 737-744.
22 Hogg K, Swedberg K, McMurray J. Heart failure with preserved left ventricular systolic function; epidemiology, clinical characteristics, and prognosis. *J Am Coll Cardiol* 2004; 43: 317-327.
23 Hernandez AF, Hammill BG, O'Connor CM, et al. Clinical effectiveness of beta-blockers in heart failure: findings from the OPTIMIZE-HF (Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure) Registry. *J Am Coll Cardiol* 2009; 53: 184-192.
24 Kotlyar E, Keogh AM, Macdonald PS, et al. Tolerability of carvedilol in patients with heart failure and concomitant chronic obstructive pulmonary disease or asthma. *J Heart Lung Transplant* 2002; 21: 1290-1295.
25 Juurlink DN, Mamdani MM, Lee DS, et al. Rates of hyperkalemia after publication of the Randomized Aldactone Evaluation Study. *N Engl J Med* 2004; 351: 543-551.

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