

this finding in the absence of formerly known causes has not been extensively studied.

Objective: To describe the characteristics and clinical outcomes associated with an abnormal LVCR.

Methods: Searched PubMed, Embase database under the guidance of a trained librarian for the keywords “left ventricle/ventricle/myocardium” and “contractile reserve/contraction/contractility” and “prognosis/outcome/mortality/survival”. We selected 27 studies that satisfied the eligibility criteria and were summarised according to PRISMA protocol describing patients’ characteristics.

Results: There were 2435 subjects (66.8% men) categorised in four subgroups according to the pathophysiological process: dilated cardiomyopathy, ischaemic cardiomyopathy, valvular disease and miscellaneous. Dobutamine stress echocardiogram was the most commonly performed modality (69%) followed by exercise stress echocardiogram (17%), dipyridamole stress echocardiogram (7%), invasive haemodynamic measurement (4%) and dobutamine stress MRI (3%). (Fig. 1) A diverse range of indices were utilised to measure LVCR including Δ LV ejection fraction (40.7%), Δ wall motion score index (33.3%), Δ stroke volume (11.1%), Δ global strain rate (7.4%), Δ cardiac power output (3.7%), Δ S’ (3.7%), Δ Fractional area (3.7%). (Fig. 1) Most studies (96.2%) demonstrated a significant correlation between the absence of LVCR and raised cardiovascular events, cardiac death and all-cause mortality.

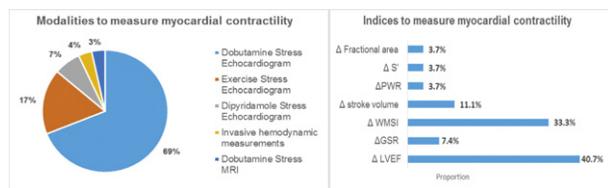


Fig. 1.

Conclusions: This study suggests an abnormal LVCR is associated with increased adverse outcome, regardless of the underlying cardiac pathology.

<http://dx.doi.org/10.1016/j.hlc.2017.06.230>

230

Rates of 30-Day Readmission and Mortality After Heart Failure Hospitalisation in Australia and New Zealand: A Population Study



C. Labroschiano^{1,2,3,*}, T. Air^{1,2,3},
R. Tavella^{1,3,4}, J. Beltrame^{1,3,4}, C. Zeitz^{1,3,4},
D. Horton^{2,5}, I. Ranasinghe^{1,2,4}

¹ The University of Adelaide, Adelaide, Australia

² Health Performance and Policy Research Unit, The Basil Hetzel Institute, Adelaide, Australia

³ Translational Vascular Function Research Collaborative, The Basil Hetzel Institute, Adelaide, Australia

⁴ Central Adelaide Local Health Network, Adelaide, Australia

⁵ Data 2 Decisions, Adelaide, Australia

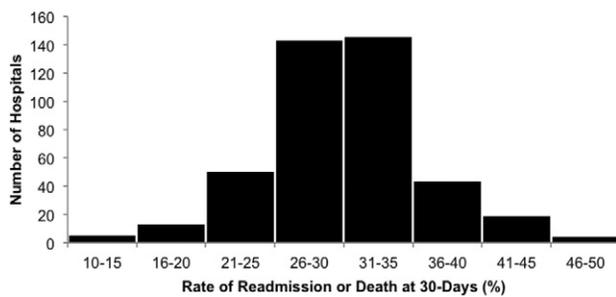
Background: Heart failure (HF) is a common cause of hospitalisation yet no national data exists on the early outcomes following a HF hospitalisation. We assessed 30-day all-cause readmission and mortality following a HF hospitalisation and how these outcomes varied among regions and hospitals.

Methods: We obtained population-wide hospitalisation data from all Australian State Health Departments (except the Northern Territory) and the New Zealand (NZ) Ministry of Health from 2010–2015 linked with Death Registries to identify post-discharge death. We identified hospitalisations with a primary discharge diagnosis of HF (ICD10AM codes I11.0, I23.0, I13.2 and I50.x). The primary study outcome was all-cause death or hospital readmission at 30 days post-discharge.

Results: We identified 219,532 HF hospitalisations where the patient was discharged alive (mean age 78.1 ± 11.9 years, 52.7% male) among 155,269 unique patients. Following discharge, 13,131 (6.0%) died and 55,018 (25.1%) were readmitted by 30-days post-discharge. Overall 67,033 (30.5%) HF hospitalisations resulted in readmission or death at 30-days. Both 30-day rates of mortality (3.7% in NZ to 7.5% in the Australian Capital Territory, $p < 0.01$) and readmission (21.9% in Queensland to 28.4% in Western Australia, $p < 0.01$) varied among regions. Overall, 432 hospitals had more than 25 HF hospitalisations ($n = 201,893$ hospitalisations) and the 30-day rates of death or readmission varied from 11.5% to 59.3% (Figure) among hospitals.

Conclusion: Thirty per cent of HF hospitalisations in Australia and NZ result in poor early outcomes with wide variation among hospitals and regions. Concerted clinical and policy intervention are needed to improve early HF outcomes.

Hospital Variation of Readmission or Death at 30-Days Post-Discharge



<http://dx.doi.org/10.1016/j.hlc.2017.06.231>

231

Refractory Cardiogenic Shock in a Young Male: Barriers to Urgent Heart Transplant



B. Moore^{1,*}, C. Hayward², A. Keech¹

¹ Department of Cardiology, Royal Prince Alfred Hospital, Sydney, Australia

² Heart Transplant Unit, St Vincent's Hospital, Sydney, Australia

A 26-year-old male presented with chest pain and an acute anterior STEMI. His past history was notable for Crohn's disease on adalimumab, as well as metallic mitral and aortic valves following *Candida parapsilosis* endocarditis on presumed rheumatic valves. Angiography revealed a saddle embolus appearance at the proximal LAD/first diagonal bifurcation, treated with two drug eluting stents. He subsequently became febrile with a suspicious echodensity seen on the mitral annulus; the embolus therefore was thought either due to culture negative recurrent prosthetic endocarditis or thrombus in the setting of a subtherapeutic INR(1.6). Empiric antibiotics were given as well as heparin anticoagulation.

His clinical course was turbulent, with refractory cardiogenic shock (ejection fraction 12%) dependent on multiple inotropes and intermittent dialysis. Concerns were raised regarding the severity of his Crohn's given intercurrent gastrointestinal bleeding; this along with suspected infection of a femoral pseudoaneurysm delayed initial transplant referral. After 4 weeks however he developed acutely worsening cardiogenic shock and was transferred to a transplant centre on VA-ECMO. Unfortunately despite urgent national listing, no body size and blood group matched donor could be found and he was delisted upon development of disseminated pulmonary aspergillosis in week 5 of VA-ECMO support. Soon after he suffered multiple embolic strokes and was palliated before passing away.

Mortality for patients in refractory cardiogenic shock remains high despite modern supportive care. Early discussion with a transplant centre regarding suitability and barriers to transplant is essential; notably intra-cardiac infection does not preclude heart transplant consideration.

<http://dx.doi.org/10.1016/j.hlc.2017.06.232>

232

Relative Effectiveness of Exercise Training, Versus Pharmacotherapies in Heart Failure with Preserved Ejection Fraction: A Meta-Analysis Focussing on Exercise Outcomes



C. Giuliano^{2,1}, R. Samuel³, R. Falls⁴, M. Woessner^{2,1}, I. Hopper⁵, S. Vogrin⁴, C. Neil^{1,4,2,*}

¹ Clinical Exercise Science Research Programme, Institute of Sport, Exercise and Active Living, Victoria University, Melbourne, Australia

² Western Health, Sunshine Hospital, Melbourne, Australia

³ Department of Cardiology, University Hospital Geelong, Geelong, Australia

⁴ Department of Medicine, The University of Melbourne, Australian Institute for Musculoskeletal Science, Melbourne, Australia

⁵ School of Public Health and Preventive Medicine, Department of Epidemiology and Preventive Medicine, Monash University, Melbourne, Australia

Background: Heart failure with preserved ejection fraction (HFpEF) is a common disorder, associated with congestive episodes, and/or symptoms attributable to heart failure. No therapy has been shown to improve long-term outcomes. However, as exertional intolerance is a major symptom burden, numerous Phase II studies have utilised exercise end-points as an indicator of benefit.

Methods: We undertook a quantitative review of studies published as of October 2016 by searching PubMed, Embase and clinicaltrials.gov, combining terms related to the population (i.e. HFpEF) with terms for the outcomes (e.g. VO_{2peak} , aerobic capacity, etc.). Random effects meta-analysis on the mean difference in raw change scores was performed with subgroup analysis for exercise and pharmaceutical studies.

Results: Five exercise and 10 pharmaceutical trials were included the meta-analysis of aerobic capacity (VO_{2peak} and 6 minute walk distance [6MWD]). For exercise studies, exercise training duration ranged from 6-16 weeks and included aerobic based regimes performed 2-3 times per week at 40-70% of VO_{2peak} . Pharmaceutical trials examined ivabradine, sildenafil, ACE-inhibition, angiotensin receptor blockade, beta-adrenergic antagonists and mineralocorticoid receptor antagonists. On average, exercise increased VO_{2peak} by 2.41 ml/kg/min, (95% CI: 1.8, 3.02) while pharmaceutical interventions did not exhibit any effect (-0.04 ml/kg/min, 95% CI: -0.64, 0.56). 6MWD was improved following exercise by 39 m (95% CI: 13, 65), but not by pharmaceutical intervention (-4 m; 95% CI: -16, 7).

Conclusion: Exercise training exhibited a strong effect on indices of functional capacity. In contrast, the pharmacotherapies tested thus far do not improve exercise tolerance.

<http://dx.doi.org/10.1016/j.hlc.2017.06.233>