**Characteristics, Management and Outcomes of Patients after an Index Heart Failure Admission**

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**Background:** Heart failure (HF) is a worldwide health problem associated with substantial in-hospital and post-discharge mortality, as well as high rates of readmission.

**Aims:** To describe patient characteristics, management and outcomes after index HF hospitalisation.

**Methods:** We extracted all index HF admissions to John Hunter and Belmont hospitals in 2014, with an ICD I50 code as a principal diagnosis.

**Results:** 289 patients were identified. 20% had HfHFrEF (EF <50%), 51% had HfHFrEF (EF ≥50%) and 28% had HFpEF (EF 34%) (Table). Of 289 patients, 46% had iron studies and 68% of those had iron deficiency anaemia. On admission, 52% of the patients were prescribed a medication associated with substantial in-hospital and post-discharge mortality, as well as high rates of readmission. 12% were on mineralocorticoid receptor (MRA) antagonists, ≥10% on ACEi/ARB, β-blocker (67%), ACEi/ARB (63%), and MRA (56%) (P = 0.001). 1-year all-cause mortality was 26%, 1-year all-cause readmission was 55% and 1-year HF readmission rate was 26%.

**Conclusion:** HF patients have multiple comorbidities and precipitants of HF decompensation. Index HF hospitalisation, irrespective of HF aetiology, was associated with significant 1-year morbidity and mortality despite prescription of guideline-directed HF therapies during that admission.

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**Clinical Outcomes for Patients with Left Ventricular Non-Compaction**

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**Background:** Left ventricular non-compaction (LVNC) is characterised by prominent LV trabeculae. The diagnosis is based on clinical and morphologic criteria but remains difficult without a gold standard. The most commonly used CMR criteria defines LVNC as an end-diastolic non-compacted to compacted ratio of ≥2.3 (Peterson, 2005).

**Methods/results:** Between 2008 and 2015 we clinically followed up 180 patients with an average age of 45.5 ± 3.3 years (59 males) that satisfied the Peterson criteria. The average follow-up time was 75 ± 23 months. Amongst this group, 69 patients had normal LV systolic function and 31 had abnormal LV systolic dysfunction (LVEF ≤55%). We evaluated the CMR scans for LV wall thickness, NC location, maximal NC/C ratio and scar. We then followed up all patients for ICD insertion, shock and death. Comparing the two groups, there was no statistically significant difference in LV wall thickness, NC/C ratio or NC location; however, the LV dysfunction group had a higher incidence of scar (p = 0.0033). ICDs and death were also higher in the LV dysfunction group (RR = 3.39, 95% CI 1.31 to 8.56 p = 0.0141 and RR = 30.3, 95% CI 1.76 to 521.47, p = 0.0006, respectively).

**Conclusion:** In patients with LVNC, the risk of ICD and death is significantly higher in those with LV dysfunction irrespective of NC/C ratio, location of NC and scar.

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**Cost Consequence Analysis for Optimising Medical Therapy in an Australian Heart Failure with Reduced Ejection Fraction (HFrEF) Cohort**

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Heart failure poses a substantive economic burden on the Australian healthcare system, with known underutilisation of evidence-based pharmacotherapy. Herewith, we estimate the annual cost of optimising pharmacotherapy for heart failure with reduced ejection fraction (HFrEF) in an Australian adult cohort across 6 metropolitan hospitals in NSW.

This retrospective cohort study applied best available data to determine the relative risk reduction in mortality and heart failure-related rehospitalisation for guideline-based medical therapies (Beta-blockers, ACE-Inhibitors, Neprilysin Inhibitors, Diuretics, Aldosterone Antagonists and Ibrabide). Our cohort comprised 26% of annual heart failure...
admissions across NSW, of which 815 met inclusion criteria for HFrEF. Cost estimates were calculated from the Pharmaceutical Benefits Scheme (PBS) data for the cheapest agent within a drug class. The cost of hospital admission for heart failure was estimated using The National Efficient Price (NEP) 2018 calculator issued by the Independent Hospital Pricing Authority (IHPA) in Australia.

Of the 815 patients that met inclusion criteria for hospital admission due to HFrEF, the annual rate of readmission for heart failure was 52%, with all cause mortality rate being 14%. We found a 77% average reduction in mortality benefit and a 71% average increase in rehospitalisation due to suboptimal pharmacotherapy. In our cohort, 28 deaths and 123 rehospitalisations could have been prevented by pharmacotherapy optimisation. The annual cost per patient of optimising pharmacotherapy was $135,35, resulting in a total projected cost of $303,874.80 to optimise HFrEF pharmacotherapy across NSW.

The gap in optimal pharmacotherapy prescription for heart failure is a major contributing factor to hospital readmissions, thus posing a significant economic burden to the NSW healthcare system. More comprehensive Australia-specific cost-benefit analyses including recommended device therapies will better elucidate this major public health burden.

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Decline in Left Ventricular Ejection Fraction in Patients Undergoing Pacemaker Implantation
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Background: RV pacing has been associated with impaired left ventricular function resulting in heart failure. Prediction of patients susceptible to this condition is important as implantation of an LV lead may prevent it.

Aim: The aim of this study was to identify clinical, ECG and echocardiographic predictors of decrease in LV function in patients undergoing pacemaker implantation.

Methods: Retrospective analysis of 106 consecutive patients with preserved LVEF receiving a pacemaker at a tertiary hospital from 2010 to 2018 with follow up echo >6 months post implantation. We stratified the patients into groups based on tercile of LVEF change.

Results: Mean ± SD age was 72.7 ± 12.6 yrs; 34.9% were female; baseline mean LVEF was 58 ± 9%. Pacing indication was sinus node diseases (39%) or AV conduction diseases (61%). After a median (25–75 percentile) follow up of 3 (1–4) yrs, LVEF decreased by 8.4 ± 11.2%, ranging from −2% to −4% (first tercile), −4 to −12% (second), and −12 to −45% (third).

Patients with greatest LVEF decrease (n = 72; 4–45% drop) were less likely to have atrial fibrillation; due to pre-implant (49% vs 79%; P = 0.003), had a higher pre-implant LVEF (60% vs 54%; P < 0.05) than patients with LVEF being no change or increase (n = 34).

Pacing indication, baseline QRS width, RV lead position, and pacing burden did not significantly differ between groups.

Conclusion: LVEF reduction occurred in 73.6% of patients undergoing pacemaker implantation for bradycardia; standard clinical, ECG and echo parameters failed to identify this group.

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Defining the Characteristics of a More Clinically Relevant Mouse Model of Type-2 Diabetes (T2D)-Induced Cardiomyopathy
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Diabetes is associated with an increased risk of heart failure due to altered structure and function of the heart, commonly termed diabetic cardiomyopathy. Mice are the most widely utilised for experimental studies investigating diabetes, however most are genetic models of diabetes (e.g. db/db, ob/ob) and do not reflect the human condition due to their altered leptin signalling. We sought to characterise the altered cardiac, kidney and liver structure and function in a mouse model of T2D-induced cardiomyopathy incorporating low dose streptozotocin (STZ) and high-fat diet.

Male 6-week-old FVB/N mice received three consecutive daily i.p. injections of STZ (55 mg/kg, body weight) followed by 26 weeks of high-fat diet (42% energy from lipids, SF04-001, Specialty Feeds) to induce T2D. Non-diabetic (ND) mice received vehicle and normal chow diet. Blood glucose (fortnightly), Doppler and tissue Doppler echocardiography, body composition, plasma insulin, markers of myocardial, kidney and liver structure, remodelling and function, were assessed.

T2D mice had significantly increased weight and fat mass, exhibited increased blood glucose and plasma insulin, and albuminuria (P < 0.05 for all). Heart weight and left ventricular weight, as well as kidney weight were unaltered with T2D, however liver and spleen weights were significantly increased (P < 0.05). Markers of fibrosis (MMP9, PAI-1), hypertrophy (β-myosin heavy chain) and apoptosis (p-P46 JNK) were increased in T2D hearts (P < 0.05 for all). At endpoint, there was clear evidence of diastolic dysfunction in terms of reduced E/A ratio and e’/a’ ratio, and prolonged deceleration time and IVRT (P < 0.05 for all). In the kidney,