sation are not well characterized. Mechanosensation has traditionally been modelled using stem cells but unlike stem cells, cardiomyocytes have muscle actomyosin and are mechanotransduced linked to adjacent cells through intercalated discs, so traditional models of mechanosensation may not be suitable for understanding cardiomyocyte mechanobiology. To better understand cardiomyocytes mechanobiology, gelatin methacryloyl (GelMA) was used to create a stiffness gradient hydrogel platform, with a linear stiffness gradient from 9 to 29 kPa. This platform was used to screen the mechanobiology of H9C2 cells. To investigate the relationship between mechanosensation and cardiomyocyte development and behaviour, the expression of the mechanomarkers (YAP and MRTF-A) were measured in relation to physical characteristics of the cell (size, shape and stiffness). Present data suggests that cell stiffness increases with substrate stiffness (Pearson’s Correlation Coefficient, $R^2 = 0.76$, $P < 0.05$) but no correlation was found between substrate stiffness and cell size, form factor, YAP expression or MRTF-A expression (Pearson’s Correlation Coefficient, $P > 0.05$).

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Lavare Cycle Does not Induce Pulsatility in HVAD Patients

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Background: Lavare cycle (LC) is a pre-programmed speed modulation algorithm (SMA) in the HeartWare continuous-flow left ventricular assist device (HVAD) designed to increase ventricular and pump washout. Whether the LC can induce artificial pulsatility or influence autonomic physiology in CF-LVAD patients remains unproven.

Methods: LC impact was prospectively assessed in a 10-patient crossover study. Blood pressure (BP) pulsatility, flow pulsatility and heart rate variability (HRV) were assessed during two 24-hour periods (LC off/on). Patients were monitored using: 1) Sphygmocor 24-hour ambulatory oscillographic/orcimetric BP monitoring to calculate pulse pressure (PP) and mean arterial pressure (MAP), 2) CDAS continuous pump data recorder to calculate a flow pulsatility index (PI), defined as (maximum − minimum flow)/(mean flow) and 3) Holter monitor (1000 Hz) to calculate time domain measures of HRV. BP flow and PI analyses were dichotomised for awake and asleep hours to assess diurnal variation.

Results: PP and MAP results were available for 9 patients, PI for 8 and HRV for 7. Results reported as [median (interquartile range)]. Heart rate was higher in the LC on [86.2 (74.0–107.1)] compared with the LC off [78.7 (71.2–103.9)] group ($P < 0.028$). There was no difference between groups in time domain measures of HRV (SDNN, RMSSD), markers of sympathetic and parasympathetic activity, respectively. Similarly, PI, MAP or PI did not differ significantly between groups. MAP was lower whilst asleep [68.8 (64.1–73.8)] than awake [78.5 (72.9–84.8)], unaffected by LC ($P = 0.004$).

Conclusion: LC did not induce meaningful artificial pulsatility or influence autonomic physiology in HVAD patients.

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Left Ventricular Ejection Fraction Improvement Post Commencement of Sacubitril/Valsartan in Heart Failure Patients

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Sacubitril/Valsartan decreases HF hospitalisation, cardiovascular mortality and all-cause mortality in patients with HF with reduced ejection fraction (HFrEF) [1]. Since addition of Sacubitril/Valsartan to Pharmaceutical Benefit Scheme in June 2017, there has been considerable uptake of this drug. The aim of this study was to assess the effect of Sacubitril/Valsartan on left ventricular ejection fraction (LVEF) in clinical practice using an intention to treat analysis in order to provide a real world experience.

Data were collected to include patients from 1st January 2017 to 31st December 2017 at a major metropolitan centre in Melbourne, Victoria via the pharmacy dispensing and clinic database. 65 patients were assessed in this study and the majority of patients had NYHA score III with pre-dominant male representation with 49 male and 16 females in the cohort. The mean age of the patients was 65.1 years (SD 12.5 years). 49% patients were diagnosed with ischaemic cardiomyopathy and 51% non ischaemic cardiomyopathy. In a 6–12 month follow-up, there was a significant improvement in LVEF from 9.7 ± 9.7% at baseline to 33.8 ± 9.9% at follow up ($P < 0.05$.

Sacubitril/Valsartan is a much needed therapeutic advancement in treatment of heart failure and supplements the current evidence-based maximal medical therapy. It provides LVEF improvement even in low doses and in a short time interval as evident in our study. However, further research is necessary to demonstrate whether these outcomes are sustained in the longer-term in the HFrEF population.

Reference


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