

$p < 0.01$) and, moreover, we also documented a higher sacubitril/valsartan prescription (67% vs 55%; $p = 0.06$) instead of lower loop diuretics use (47% vs 35%, $p = 0.05$). Mineralocorticoid receptor antagonist (77% vs 83%; $p = 0.19$) and beta blockers (94% in both groups) therapy did not change. With a total median follow-up of 19 months (Q1-Q3: 9–36), Kaplan-Meier mortality analysis was represented in Figure 1.

Conclusions: In our centers, since SGLT2-i approval for HFREF, there exists a fast joining of SGLT2-i to HFREF therapy. It could help us to accomplish lower rates of loop diuretics prescription enabling a better titration of drugs with proven positive impact in remodelling and morbimortality. These results support in real-world the provider role of SGLT2-i, explaining a higher increase of left ventricular ejection fraction in post SGLT2-i group, with a non-statistically significant trend to lower mortality probably due to a small follow-up.

	Onset prior to introduction of SGLT2 inhibitors (n=219)	Onset after the introduction of SGLT2 inhibitors (n=95)	p
Sex (Male)	70%	77%	0,206
Age (years)	64 ± 13	63 ± 14	0,858
Hypertension	68%	60%	0,229
Diabetes	36%	30%	0,347
Previous coronary percutaneous intervention	19%	25%	0,279
COPD	20%	17%	0,604
GFR (ml/min)	74 ± 22	69 ± 24	0,181
Total Bilirubin (mg/dl)	0,77 ± 0,53	0,78 ± 0,44	0,379
Basal NTproBNP (pg/ml)	1858 (1012 – 3340)	2252 (1015 – 5195)	0,270
Atrial Fibrillation	36%	38%	0,758
LBBB	30%	32%	0,693
Basal LVEF (%)	31 (25-36)	30 (25-36)	0,519
Basal LVEDD (mm)	58 ± 10	60 ± 12	0,334
Basal RV dysfunction	31%	33%	0,639
NYHA III/IV at follow-up*	12%	8%	0,454
LVEF (%) at follow-up*	40 (33-47)	44 (35-50)	0,015
LVEF difference (%) between basal and follow-up	9 (1-18)	12 (5-24)	0,019

Follow-up clinical visit

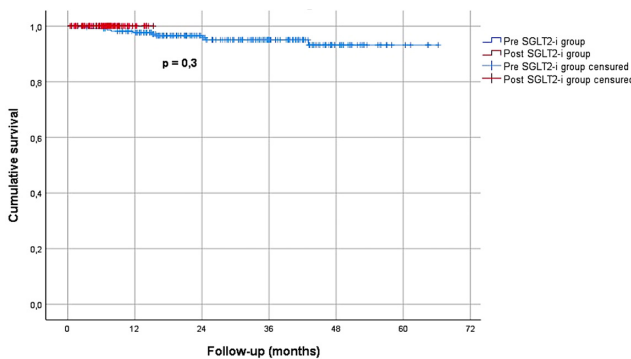


Figure 1. Kaplan-Meier mortality analysis.

Figure 1.

Impact of heart failure evidence-based-therapy in hypotensive patients

M Ana Fonseca¹; C Grijo¹; A Costa¹; C Reis¹; R Ribeiro¹; F Correia¹; A Toste¹; C Guimaraes¹; R Gouveia²; C Elias¹; I Matos¹; M Carreira¹; J Pereira¹; J Almeida¹; P Lourenco¹; ¹Sao Joao Hospital, Porto, Portugal;

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Background: Patients with hypotension have consistently been excluded from heart failure (HF) randomized controlled trials. This group of HF patients is largely unstudied. We aimed to characterize HF patients with hypotension.

Methods: We retrospectively studied adult outpatients with systolic dysfunction followed in our HF clinic from January 2012 to December 2020. Patients without blood pressure measurement on the index visit (first medical visit) were excluded. We defined hypotension as systolic blood pressure (SBP) of less than 100 mmHg. The endpoint under analysis was all-cause mortality. Patients were followed until January 2023. Patients with hypotension were compared with the remaining. A Cox-regression analysis was used to assess the prognostic impact of hypotension and to study the prognostic impact of evidence-based therapy separately in HF patients with SPB < 100mmHg and those with SPB ≥ 100mmHg. Adjustments were made considering potential confounders.

Results: We studied 1206 chronic ambulatory HF patients, 64.9% male, mean age 71 years, 47.4% with severe systolic dysfunction. Regarding the medication in use, 91.4% were on beta blockers (BB), 82.8% were on renin-angiotensin system inhibitors (RASi), including angiotensin converting enzyme inhibitors, angiotensin receptor blockers or angiotensin receptor neprilysin inhibitors; 29.6% were on mineralocorticoid receptor antagonists (MRA). A total of 157 patients (13.0%) presented SBP < 100mmHg on the index visit. Hypotensive patients more often presented atrial fibrillation and severe systolic dysfunction; they had lower haemoglobin values and higher brain natriuretic peptide (BNP) levels. Patients with hypotension were less medicated with RASi (70.7% vs 84.6%, $p < 0.001$) but more with MRA (39.6% vs 28.1%, $p = 0.004$) and diuretics (86.6% vs 78.6%, $p = 0.02$). There were no differences regarding BB use between both groups. During a median follow-up of 47 (27–85) months 645 (53.5%) patients died, 61.1% in those with hypotension and 52.3% in the remaining, $p = 0.04$.

The use of RASi in hypotensive patients was associated with better survival (HR = 0.42 (0.26–0.69)) as in those with SPB ≥ 100mmHg (HR = 0.64 (0.51–0.80)). Contrarily to patients with SPB ≥ 100mmHg, in those with hypotension, BB use was not associated with survival benefit (HR = 0.61 (0.46–0.81) and 0.98 (0.48–1.97), respectively). MRA use showed no prognostic impact in either group.

Conclusions: Hypotension was associated with poor prognosis in HF patients. In HF patients with SBP < 100mmHg, BB and MRA use did not impact prognosis, however, RASi use portended a survival benefit. Despite their exclusion from most HF therapy trials, hypotensive patients might benefit from RASi drugs.

Renal and cardiac effects of salt loading in ambulatory heart failure patients

NCT04226755 J Dauw¹; E Meekers¹; P Martens¹; P Nijst¹; L Mesotten¹; W Marchal²; M Dupont¹; W Mullens¹; ¹Hospital Oost-Limburg (ZOL), Genk, Belgium; ²Hasselt University - transnational University Limburg, Diepenbeek, Belgium;

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Background: Current guidelines recommend to limit sodium intake in heart failure (HF) patients. However, stringent sodium restriction can increase neurohormonal activation, decrease quality of life and was not advantageous in recent trials. In addition, recent studies suggest that the skin can function as a sodium buffer.

Purpose: To study effects and handling of an increased salt load in patients with HF and reduced ejection fraction.

Methods: Eighteen patients with HF and left ventricular ejection fraction < 40% and 10 age- and sex-matched healthy volunteers without cardiovascular disease were included. HF patients with severe right ventricular dysfunction, eGFR < 30 mL/min/1.73 m² or severe valvular dysfunction were excluded. After 2 weeks of run-in, all study participants received 3 grams of sodium chloride (capsules of 1 g three times daily) on top of their usual diet for 4 weeks. Patients were evaluated at inclusion, at 2 weeks (end of run-in), 4 weeks (2 weeks of sodium chloride intake) and 6 weeks (4 weeks of sodium chloride intake). At each evaluation, clinical parameters, Everest congestion score, lab, echocardiography, 24-hour urine collection and bio-impedance measurements of total body water were performed. Blood volume and plasma volume were assessed using a radio-labeled red blood cells dilution technique before salt loading (at 2 weeks) and at the end of the study (at 6 weeks). At the same time points, a skin biopsy was taken at the lower leg to assess skin sodium content and glycosaminoglycan content.

Results: Mean age was 66 ± 8 years, 2 (11.1%) were female, median LVEF was 35 (31–39) %, median eGFR was 68 (51–74) mL/min/1.73 m² and median NT-proBNP was 431 (275–961) ng/L at baseline and all patients were optimally treated medically. Salt loading did not influence weight, blood pressure, congestion score or NT-proBNP (Figure 1). There was no significant change in total body water (from 46.87 L to 44.41 L; $p = 0.780$), plasma volume (2735 mL vs. 2904 mL; $p = 0.231$) and total blood volume (4748 mL vs. 4885 mL; $p = 0.327$). Renal sodium excretion increased from 150 ± 55 mmol/24h to 173 ± 58 mmol/24h ($p = 0.024$), while plasma renin decreased from 286 (25–550) μU/L to 88 (19–362) μU/L ($p = 0.002$) (Figure 2). Salt loading did not significantly influence LVEF (from 35% to 35%; $p = 0.801$), left

ventricular end-diastolic volume indexed (from 111 mL/m² to 115 mL/m²; $p = 0.085$) and E/e' (from 10 to 10; $p = 0.559$). There was no significant change in skin sodium content (3.64 vs. 3.91 mg/g dry weight; $p = 0.265$), total glycosaminoglycan content (14.76 vs. 15.45 $\mu\text{g/g}$ dry weight; $p = 0.415$) or sulfated glycosaminoglycan content (5.83 vs. 5.86 $\mu\text{g/g}$ dry weight; $p = 0.880$). Results were similar in healthy volunteers. **Conclusion:** Salt loading is well tolerated in selected patients with HF_{rEF}, compensated by increased renal sodium excretion and associated with decreased neuro-hormonal activation.

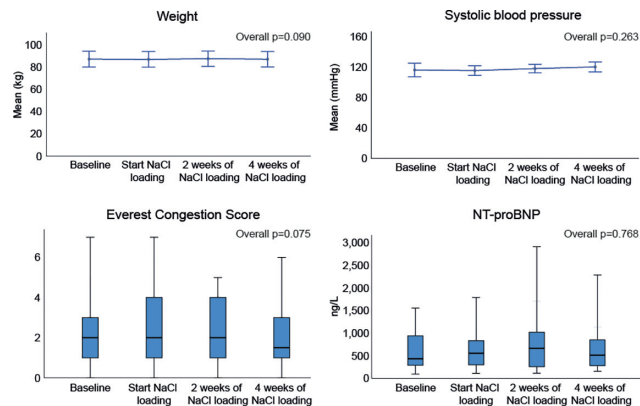


Figure 1

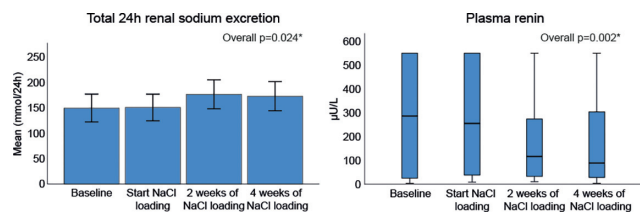


Figure 2

Different cardioprotection protocols for elective heart surgery surgery in patients with advanced left ventricular failure- a comparison of del Nido and cold blood cardioplegia.

K Sanetra¹; W Gerber¹; M Mazur²; M Synak²; M Kubaszewska²; E Pietrzyk²; PP Buszman¹; J Jankowska-Sanetra¹; M Kachel¹; K Milewski¹; P Kazmierczak¹; A Bochenek¹; ¹American Heart of Poland, Katowice, Poland; ²Andrzej Frycz Modrzewski Krakow University, Krakow, Poland;

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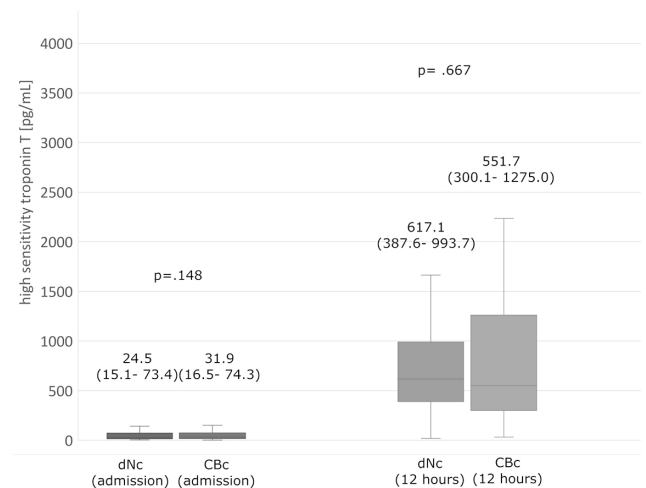
Objective: To determine the efficacy of the del Nido cardioplegia in patients with severely impaired ejection fraction undergoing elective surgery.

Methods: Institutional database was searched for patients with severely reduced ejection fraction ($\leq 35\%$). Further, patients who received del Nido (dNc) or cold blood cardioplegia (CBc) were selected. Subjects who underwent emergency surgery were excluded. Propensity matching was performed based on age, gender and number of conducted procedures. Observation towards changes in ejection fraction and postoperative troponin release at 12 hours was performed. Other endpoints included: mortality, stroke, perioperative myocardial infarction, composite endpoint (MACCE) and other postoperative complications.

Results: Propensity matching revealed 161 pairs of patients. The preoperative ejection fraction in both groups was similar (median; iQR: 30%; 25–35% vs 30%; 25–35%; $p = .771$). There were no other differences at baseline. In both groups, similar number of patients underwent each kind of surgery. Perioperatively, there was no difference in mortality (3.7% vs 1.2%; $p = .151$), occurrence of myocardial infarction (4.9% vs 3.7%; $p = .585$), stroke (1.2% vs 2.5%; $p = .411$) or MACCE (9.3% vs 6.2%; $p = .298$). Postoperative troponin release at 12 hours was similar (median, IQR: 617.1, 387.6- 993.7 pg/ mL vs 551.7, 300.1- 1275.0 pg/mL; $p = .667$). However, lower number of patients in dNc group had significant (>5%) postoperative fall in ejection fraction (1.2% vs 6.2%; $p = .019$).

Conclusions: Both protocols provide satisfactory cardioprotection in patients with heart failure undergoing cardiac surgery. The preservation of ejection fraction is

greater in patients who receive del Nido cardioplegia. Further, prospective studies are required.



Troponin T values in time intervals.

18-month pattern of cardiac-specific biomarkers and replacement and interstitial fibrosis in dilated cardiomyopathy

S Sylwia Wisniowska-Smialek¹; M Szymanska²; E Wypasek³; E Dziewiecka¹; L Vashchelina¹; P Banyas²; M Urbanczyk- Zawadzka²; M Krupinski²; G Wasilewski¹; I Gorkiewicz-Kot¹; K Wierzbicki¹; P Rubis¹; ¹Jagiellonian University Medical College, John Paul II Hospital, Krakow, Poland; ²John Paul II Hospital, Krakow, Poland; ³John Paul II Hospital, Center for Medical Research and Technology, Krakow, Poland;

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Introduction: Troponin T (TnT) and N-terminal proBNP (NT-proBNP) are established cardiac-specific biomarkers in heart failure (HF) and dilated cardiomyopathy (DCM). Cardiac magnetic resonance (CMR)-derived replacement and interstitial fibrosis are DCM hallmarks. There is scarce of data regarding the patterns of TnT and NT-proBNP and their relations with fibrosis in DCM.

Methods: Between May 2019 and September 2020, 99 DCM patients (88 male, mean age 45.2 ± 11.8 years, mean EF $29.7 \pm 10\%$) underwent CMR and serial (baseline, 3, 6, 9, 12 and 18-month) measurements of TnT and NT-proBNP. Replacement fibrosis was assessed with late gadolinium enhancement (LGE), whereas interstitial

Fig. 1. 18- month kinetic profiles of TnT and NT-proBNP levels.

