($r=0.32$ and $r=0.24$ respectively; $P<0.001$). There was extreme co-linearity between D-PVS and haemoglobin in linear regression models with other continuous variables ($VIF = 14$). Higher H-PVS (hazard ratio (HR) = 1.01 (95% confidence interval (CI) = 1.00 – 1.02); $P=0.002$) or D-PVS (HR = 1.08 (95% CI = 1.03 – 1.14); $P=0.002$) was associated with greater risk of all-cause mortality or hospitalisation with heart failure. Neither the Harrel's C-statistic nor AIC of outcome models for 1 year mortality including either weight or haemoglobin were improved by the addition of H-PVS or D-PVS (figure 2).

Conclusions Changes in weight or haemoglobin in patients with CHF are not always due to changes in plasma volume. Despite apparent associations with disease severity, H-PVS and D-PVS are just surrogates of the variables from which they are calculated and not reliable measures of congestion. It is likely neither has any clinical utility.

Conflict of Interest None

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A SINGLE CENTRE STUDY ASSESSING THE EFFECTIVENESS OF ACTIVE MONITORING, USING TELEMEDICINE, IN PREVENTING HEART FAILURE ADMISSIONS AND DEATH IN A PRIMARY CARE POPULATION WITH SYSTOLIC HEART FAILURE

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Introduction Patients with systolic heart failure are at high risk of admission to hospital and death. This can be reduced by ensuring that they are receiving all evidence-based heart failure medications and by detecting early signs of deterioration in their condition.

Methods Patients were enrolled from 2016 through 2017 at 12 GP practices across Buckinghamshire. Practices were selected if they used EMIS web as their electronic patient record and showed enthusiasm for participating in the study. At each practice, a senior heart failure nurse was deployed to identify patients with a REED code for heart failure and an echocardiographically determined ejection fraction (EF) of less than 40%. If no echocardiogram was available this was arranged. We recruited 209 primary care patients with echocardiographically proven left ventricular systolic dysfunction (ejection fraction <40%). 84 patients consented to be actively monitored by the heart failure team using telemedicine. It automatically uploaded any relevant data (weight, dyspnoea class, renal function, full blood count, urate, chest X-ray, repeat echocardiogram, hospital admissions, death) entered in the GP or hospital records. Patients were also encouraged to enter their own data (in particular weight and exercise tolerance). Clinicians were instructed to treat patients in accordance with national guidelines for the management of heart failure. 125 patients consented to receiving usual care but allowing access to their medical records. The primary end-point was cardiovascular death or admission to hospital for heart failure at 1 year. Secondary end-points included the prescription of evidence-based heart failure medications and patient satisfaction at the end of the study.

Results There was no difference in the mortality rate between the groups (6.02% in the active group and 5.56% in control). There was a significant difference in hospital admission (10.84% in the active group and 1.59% in control; p -value of 0.0078). At the end of the study, in the active group v control group, 92% v 52% of patients were on a beta-blocker, 92% v 48% on ACE-I/ARB, and 60% v 30% on an MRA. There were no differences in the final doses achieved.

Conclusions Active telemonitoring in an elderly population with systolic heart failure did not reduce cardiovascular mortality or admission to hospital for heart failure over the 1 year of the study. It did result in more patients receiving evidence-based heart failure medications. Overall satisfaction with active monitoring delivery a questionnaire was provided to all patients who opted to receive active monitoring. The purpose of the questionnaire was to determine overall satisfaction levels with the monitoring and identify areas for potential improvement. A questionnaire was circulated at the midpoint of the study (6 months) as well as at its conclusion (12 months). Of all patients in the active control group, a total of 27 completed both questionnaires (corresponding to 32.53% of all patients under active monitoring). The characteristics of this subset of the active cohort were consistent with the overall characteristics of all patients in the study; there was no significant differences in cardiovascular status (e.g., NYHA scores) or personal characteristics (e.g., gender, age).

The questionnaire consisted of eleven questions measuring the patient satisfaction on various aspects of the active monitoring treatment. The overall response from patients was positive, with an average satisfaction of 8.54 / 10. Furthermore, when asked how likely they would be recommend the active monitoring treatment, patients gave an average score of 8.34 / 10.

Conflict of Interest None

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SIDE EFFECTS OF SACUBITRIL/VALSARTAN INFLUENCE OUTCOMES IN HEART FAILURE POPULATIONS: A RETROSPECTIVE STUDY

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Introduction Sacubitril/Valsartan (SV) is an effective treatment for patients with heart failure and reduced ejection fraction (HFrEF), decreasing hospitalisation and reducing the overall the risk of cardiovascular death.¹ In the PARADIGM study a higher rate of hypotension and nonserious angioedema was found in the SV vs enalapril arm, but a lower incidence of renal impairment, hyperkalaemia and cough. Until now, there is little data on tolerance of SV outside this clinical trial as well as the consequence of the discontinuation of this therapy. We therefore analysed the side effects of SV in a real-world practice and investigated the outcomes of stopping SV.

Methods We conducted a retrospective analysis of consecutive patients with heart failure and reduced ejection fraction who were started on SV at University Hospitals Coventry and Warwickshire NHS Trust between March 2017 and July 2020. All

data points were retrieved from the hospital database up to March 2021.

Results A total of 147 patients were analysed with a mean follow-up of 27.2 ± 8.8 months (table 2). 17 (12%) patients stopped SV (table 1). Those who stopped SV were more likely to be hospitalised for heart failure ($n=5$ [29.4%] vs $n=8$ [6.9%]; $p<0.01$) or die ($n=7$ [41.1%] vs $n=8$ [6.2%]; $p<0.01$) when compared to those continuing SV (table 2). Months to hospitalisation and death in those who stopped SV were 11.0 ± 12.7 and 9.9 ± 10.8 respectively (table 2). When SV was stopped, 10 (58.8%) started an angiotensin-2 converting enzyme inhibitor (ACEi), 1 (6.0%) an angiotensin-2 receptor blocker (ARB), 4 (23.5%) stopped treatment 1 (6.0%) a nitrate and 1 (6.0%) digoxin in place of SV (table 2). The maximum dose was reached in 44 (30%) of the cohort up to point of data collection. Renal impairment ($n=20$ [13.6%]) and postural hypotension ($n=19$ [12.9%]) were the most

common reported side effects (table 2). Reasons for stopping SV were most commonly renal impairment ($n=7$ [41%], creatinine increase of 30.6 ± 51.7 at point of stopping entresto), headache/dizziness ($n=4$, [23.5%]) and postural hypotension ($n=3$ [17.6%]) (table 2).

Discussion In this real-world retrospective study we could demonstrate that discontinuation of SV is associated with a higher chance of hospitalisation for heart failure and death. 17 (11.5%) patients stopped SV all of which had side effects and 7 (41.2%) died, though the characteristics of patients who stopped SV were older age, male, worse renal function prior to SV and higher incidence of atrial fibrillation (table 1). In the PARADIGM study the number adverse events was 2247 (rate not available) and 76 (1.8%) patients discontinued SV. Compared to our data discontinuation of SV is much higher possibly due to the cohort in the PARADIGM-HF trial all tolerating max dose initially. We suggest further prospective

Abstract 144 Table 1 Demographics and baselines characteristics

	SV Continued n=130	SV Discontinued n=17	P
Demographics:			
Age (mean)	67.1 ± 12.8	72.0 ± 6.2	<0.01
Female (%)	40; (30.8)	1; (5.9)	0.04
Ethnicity:			
White n; (%)	111; (85.4)	17; (100)	0.42
Asian n; (%)	8; (6.1)	0	
Black n; (%)	6; (4.6)	0	
Other n; (%)	5; (3.8)	0	
Comorbidities:			
Hypertension n; (%)	61; (46.9)	9; (52.9)	0.64
Diabetes n; (%)	37; (28.4)	5; (29.4)	1.00
Ischaemic Heart Disease n; (%)	60; (46.2)	5; (29.4)	0.19
Chronic Kidney Disease n; (%)	9; (6.9)	2; (11.8)	0.62
AF n; (%)	35; (26.9)	9; (52.9)	0.03
Stroke n; (%)	9; (6.9)	0	0.60
Baseline Measures:			
Systolic Blood Pressure	130.5 ± 19.5	122.4 ± 17.1	0.49
Diastolic Blood Pressure	83.6 ± 22.1	78.1 ± 9.6	0.20
NYHA Class	2.5 ± 0.6	2.5 ± 0.5	
Creatinine	100.7 ± 21.9	115.1 ± 41.0	<0.01
GFR	55.8 ± 7.2	53.8 ± 11.3	<0.01
Left Ventricular Ejection Fraction %	26.3 ± 8.5	26.5 ± 7.6	0.56
Baseline Medications:			
Diuretic n; (%)	91; (70.0)	11; (64.7)	0.66
ACEi/ARB n; (%)	120; (92.3)	16; (94.1)	0.79
Beta-blocker n; (%)	116; (89.2)	16; (94.1)	0.53
MRA n; (%)	52; (40.0)	7; (41.1)	0.93
AF: Atrial Fibrillation, GFR: estimated Glomerular Filtration Rate, ACEi: Angiotensin Converting Enzyme inhibitor, ARB: Angiotensin II Receptor Blocker. MRA: Mineralocorticoid Receptor Antagonist			

AF: Atrial Fibrillation, GFR: estimated Glomerular Filtration Rate, ACEi: Angiotensin Converting Enzyme inhibitor, ARB: Angiotensin II Receptor Blocker, MRA: Mineralocorticoid Receptor Antagonist

Abstract 144 Table 2 Outcomes and side effects

	SV Continued n=130	SV Discontinued n=17	P
Outcomes:			
Mean Follow-up (months)	27.2 ± 9.0	27.3 ± 7.5	0.13
LVEF Improvement ≥ 10% n; (%)	37; (28.4)	2; (11.8)	0.42
Hospitalisation n; (%)	8; (6.1)	5; (29.4)	<0.01
Death n; (%)	8; (6.1)	7; (41.1)	<0.01
SV Stop to Hospitalisation in Months	-	11.0 ± 12.7	-
SV Stop to Death in Months	-	9.9 ± 10.8	-
Post SV Measures:			
GFR Change	-4.6 ± 10.2	-7.4 ± 12.7	0.23
LVEF %	39.5 ± 13.5	35.8 ± 14.0	0.71
Side Effects:			
Renal Impairment n; (%)	13; (10.0)	7 (41.1)	<0.01
Hyperkalaemia n; (%)	4; (2.5)	0	1.00
Headache/Dizziness n; (%)	9; (6.9)	4; (23.5)	0.05
Postural Hypotension n; (%)	16; (12.3)	3; (17.6)	0.46
Cough n; (%)	1; (0.8)	4; (23.5)	1.00
Shortness of Breath n; (%)	1; (0.8)	2; (11.8)	0.04
Diarrhoea n; (%)	1; (0.8)	2; (11.8)	0.04
Skin Irritation n; (%)	0	2; (11.8)	0.01
Fatigue/Nausea n; (%)	6; (4.6)	2; (11.8)	0.23
Medication after stopping SV:			
No Drug n; (%)	-	2; (11.8)	-
ACEi n; (%)	-	10; (58.8)	-
ARB n; (%)	-	1; (5.9)	-
Digoxin n; (%)	-	1; (5.9)	-
Nitrate n; (%)	-	1; (5.9)	-

LVEF: Left Ventricular Ejection Fraction

investigation on how to improve maintain/titrate to max dose SV in cohorts of HFrEF patients who do not tolerate SV well with respect to outcomes.

Conflict of Interest Nil

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INVESTIGATING THE DIRECT IMPLICATIONS OF THE DAPA HF TRIAL AND THE INTRODUCTION OF SODIUM-GLUCOSE LIKE CO-TRANSPORTER 2 (SGLT2) INHIBITORS ON MIDDLESBROUGH'S HEART FAILURE POPULATION

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Background DAPA HF was an international, multicentre, parallel group, event-driven, randomized, double-blind, placebo-controlled study in patients with chronic heart failure with

reduced ejection fraction (HFrEF), evaluating dapagliflozin versus placebo. This showed individuals with Heart Failure with Reduced Ejection Fraction (HFrEF) (NYHA II–IV, LVEF ≤40%) with or without Type 2 Diabetes Mellitus (T2DM), addition of the SGLT-2 inhibitor dapagliflozin decreased rates of cardiovascular death or worsening heart failure (NNT=21), reduced hospital admissions at 28 days (NNT=27) and all-cause mortality (NNT=43.5). We aim to ascertain the impact the introduction of SGLT2 inhibitors, could have on our local population.

Method A retrospective review of James Cook University Hospitals heart failure database (all patients with LVEF ≤ 40%) from 7/3/2019– 7/3/2020. Data recorded: Age, Diabetic Status, Renal Function, HbA1c, HFrEF medication, Diabetes Medication, Heart Failure aetiology. Exclusions: Type 1 Diabetes Mellitus, Deceased, Incomplete data. Data was reviewed by thematic analysis.

Results N=321. Mean Age- 70 years ± 10.5. Non-Diabetic / T2DM = 205/105 (64.2%/35.2%). Heart Failure aetiology: Ischaemic 137 (42.68%), Non-Ischaemic 146 (45.48%), Unknown 38 (11.83%). Renal Function: eGFR<30 mlmin⁻¹= 14, <45 mlmin⁻¹= 55 and <60 mlmin⁻¹= 131. 15 (13%) T2DM patients are on SGLT2 inhibitors. 0 Non-Diabetic

Correction: 144 Side effects of sacubitril/valsartan influence outcomes in heart failure populations: a retrospective study

Kumar S, He H, Kuehl M, *et al.* 144 Side effects of sacubitril/valsartan influence outcomes in heart failure populations: a retrospective study. *Heart* 2021;**107**:A109-A111. doi: 10.1136/heartjnl-2021-BCS.141

This Abstract has been corrected since it was first published. The authors should have been listed in the following order: Sheethal Kumar, Hejie He, Hesham Aggour, Faizel Osman, Thomas Lachlan, Michael Kuehl.

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