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RESEARCH ARTICLE

Measuring congestion with a non-invasive monitoring device in heart failure and haemodialysis: CONGEST-HF

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Aims We examined the effectiveness of a novel cardiopulmonary management wearable sensor (worn for less than 5 mins) at measuring congestion and correlated the device findings with established clinical measures of congestion.

Methods and results We enrolled three cohorts of patients: (1) patients with heart failure (HF) receiving intravenous diuretics in hospital; (2) patients established on haemodialysis, and (3) HF patients undergoing right heart catheterization (RHC). The primary outcomes in the respective cohorts were a Spearman correlation between (1) change in weight and change in thoracic impedance (TI) (from enrolment, 24 h after admission to discharge) in patients hospitalized for HF; (2) lung ultrasound B-lines and volume removed during dialysis with device measured TI, and (3) pulmonary capillary wedge pressure (PCWP) and sub-acoustic diastolic, third heart sound (S3) in the patients undergoing RHC. A total of 66 patients were enrolled. In HF patients ($n = 25$), change in weight was correlated with both change in device TI (Spearman correlation [r_{sp}] $= -0.64$, $p = 0.002$) and change in device S3 ($r_{sp} = -0.53$, $p = 0.014$). In the haemodialysis cohort ($n = 21$), B-lines and TI were strongly correlated before ($r_{sp} = -0.71$, $p < 0.001$) and after ($r_{sp} = -0.77$, $p < 0.001$) dialysis. Volume of fluid removed by dialysis was correlated with change in device TI ($r_{sp} = 0.49$, $p = 0.024$). In the RHC cohort ($n = 20$), PCWP measured at one time point and device S3 were not significantly correlated ($r_{sp} = 0.230$, $p = 0.204$). There were no device-related adverse events.

Conclusions A non-invasive device was able to detect changes in congestion in patients with HF receiving decongestion therapy and patients having fluid removed at haemodialysis. The cardiopulmonary management device, which measures multiple parameters, is a potentially useful tool to monitor patients with HF to prevent hospitalizations.

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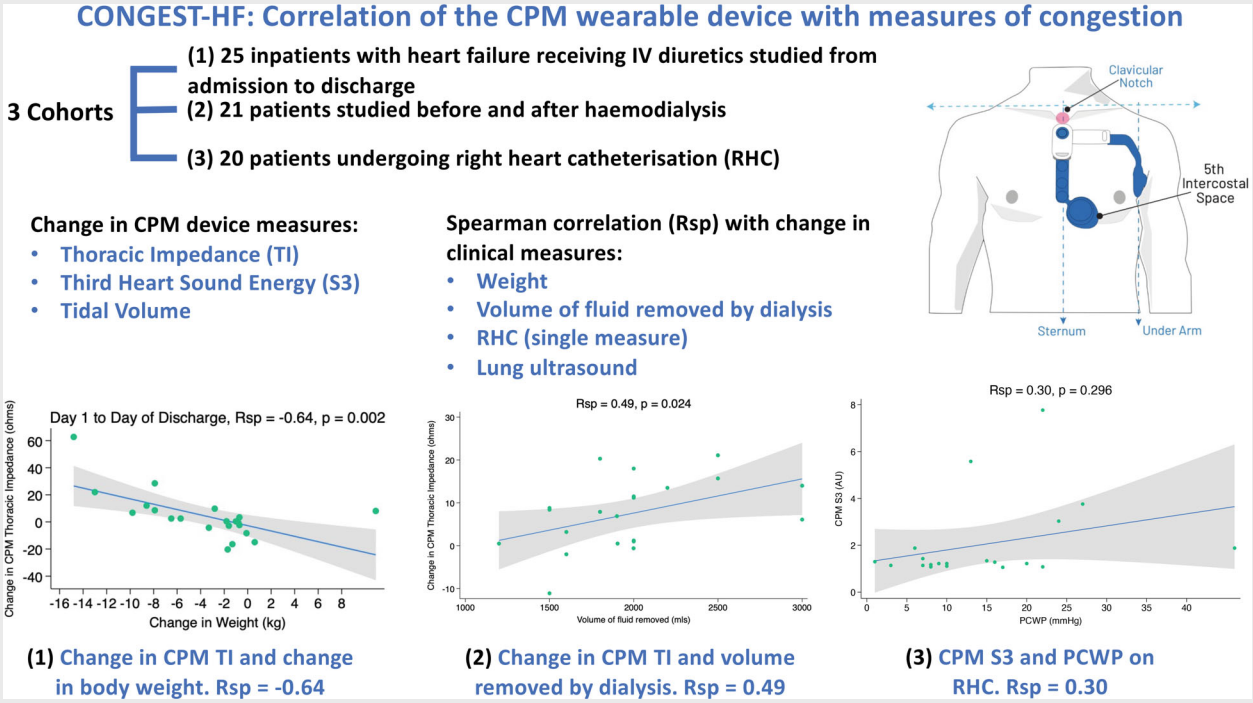
Graphical Abstract



CONGEST-HF: correlation of the cardiopulmonary monitoring (CPM) wearable device with measures of congestion. IV, intravenous; PCWP, pulmonary capillary wedge pressure.

Keywords Heart failure • Haemodialysis • Right heart catheterization • Congestion • Monitoring

Introduction

Patients with heart failure (HF) are admitted to hospital many times during their disease[1,2] for treatment with intravenous (IV) diuretics for the relief of congestion. Although there are protocols to provide IV diuretic therapy in the outpatient setting,[3] preventing hospitalizations through the early detection of deterioration and identification of subclinical congestion is preferable, allowing alteration of therapy and preventing hospitalization.

Methods to detect congestion rely on expensive invasive monitoring through functions in specially designed pacemakers[4,5] or monitoring of pulmonary artery pressure through a sensor implanted directly in the lung. Pulmonary artery pressure monitoring has been shown to reduce hospitalizations[6] but the therapy remains expensive. Simpler measures such as patient-recorded daily weight are recommended by guidelines[7] but are insensitive.[8] Wearable devices represent a potential new approach to monitor patients with HF.

We tested a non-invasive cardiopulmonary monitoring (CPM) device designed to monitor patients with HF. The wearable device is applied to the skin of the patient's chest and connects via Bluetooth to a mobile App to initiate a reading and to record physiological data. The device is worn for less than 5 min twice a day removing the need for continuous wear or invasive procedures. The CPM device uses several sensors to detect changes in thoracic impedance, a sub-acoustic diastolic, third heart sound (S3), as well as respiratory rate and tidal volume. Changes in these parameters might identify developing congestion allowing action to be taken before a patient presents with decompensation and avoiding a hospitalization. We aimed to determine if the device could detect changes in fluid status in patients with decompensated HF receiving decongestion therapy in hospital, in patients before and after a session of haemodialysis and the correlation of the measures obtained from the device with invasive measures derived from a right heart catheterization (RHC).

Methods

Study design and population

The COrrelation of the Non-invasive CardioPulmonary Management wearable device with measures of conGESTion in Heart Failure (CONGEST-HF, NCT05026034) study was a prospective, observational study designed to examine the effectiveness of the CPM

wearable device (Analog Devices Inc. [ADI], Wilmington, MA, USA) at detecting congestion and changes in congestion in three cohorts of patients. The first cohort were patients admitted to the Queen Elizabeth University Hospital (QEUH), Glasgow, UK with a diagnosis of HF and receiving IV diuretics to relieve congestion. Patients were identified early into their admission by their responsible medical teams as being volume overloaded with a primary diagnosis of decompensated HF and were then approached by the investigators. Left ventricular ejection fraction at baseline was not used to determine study eligibility. They were assessed at four time points; on the day of enrolment, the following day while still on IV diuretics, on the day of first dose of oral diuretics, and the day of discharge. The decisions to switch from IV to oral diuretics or discharge patients were made by the usual care teams independently of any information collected for the study. The second cohort were patients who had chronic end-stage renal failure and were established on haemodialysis at the QEUH for at least 90 days with a target volume removal of at least 1.5 L in a dialysis session. Fluid was removed via an indwelling catheter or arteriovenous fistula. They underwent study assessment immediately before and after a single dialysis session. The final cohort were patients undergoing a clinically indicated RHC as part of their cardiac transplant evaluation at the Scottish National Advanced Heart Failure Unit. This cohort underwent study assessment at one time point, on the day of the RHC. Full inclusion and exclusion criteria are detailed in online supplementary *Table S1* and a flow chart detailing the screening and recruitment process is given in online supplementary *Figure S1*.

Investigational medical device

The CPM device is a wearable, battery-operated, class 2a medical device. The CPM device is applied to the skin of the patient's chest, with adhesive islands, one to the left of the sternum, one at the apex of the heart, and one in the mid-axillary line (*Figure 1*). Each participant was uniquely matched to a single device for the duration of their study participation. The device communicated via Bluetooth with a mobile App which guided the investigator through a device reading. A single reading consisted of two measurements, performed whilst sitting up, then the most supine position tolerated. Each measurement lasted approximately 60 s. The CPM device measured raw data signals that were transferred by the site investigator at the time of the assessment to a secure portal managed by the Glasgow Clinical Trials Unit at the Robertson Centre for Biostatistics (RCB). ADI then downloaded the raw data and processed them into derived measurements which were returned securely to the RCB's portal. The investigators (and clinical team) were blinded to the derived device data and ADI were blinded to the investigator-collected clinical study data. The derived CPM device measurements included thoracic impedance, S3 energy and tidal volume.

Clinical study procedures

For all cohorts, at each study assessment, before device readings, an 8-zone lung ultrasound (LUS), tidal volume by bedside spirometry (Pneumotrac, Vitalograph, UK), echocardiogram (Vivid E90 for the RHC cohort and a Canon Aplio i900 for the patients hospitalized for HF or receiving dialysis; performed by an investigator [JPC] with European Association for Cardiovascular Imaging accreditation), physical symptoms and signs (including the EVEREST clinical congestion score [ECCS] comprising an assessment of dyspnoea, orthopnoea, fatigue, jugular venous distension, oedema, and pulmonary rales) and observations were recorded. Whole blood was collected at each visit for analysis of N-terminal pro-B-type natriuretic peptide (NT-proBNP) (e411, Roche Diagnostics, UK) and mid-regional pro-adrenomedullin (BRAHMS Kryptor, Thermofisher ThermoFisher Scientific, UK). All other blood samples, including haematocrit, were collected and analysed as standard care in the local hospital laboratory.

Study outcomes

The primary outcomes by cohort were: in hospitalized patients with HF, the correlation between CPM device thoracic impedance and CPM device S3 and LUS B-lines and change in weight; in the haemodialysis cohort, the correlation between CPM device thoracic impedance and both LUS B-lines and volume of fluid removed by dialysis; in the RHC cohort, the correlation between CPM device S3 and pulmonary capillary wedge pressure (PCWP) on RHC.

Secondary outcomes included the correlation of CPM device measured tidal volume with tidal volume measured by spirometry; the correlation of device measures with echocardiography, physical signs and symptoms of congestion and, in the RHC cohort only, RHC parameters additional to PCWP.

Statistical analysis

Analyses were performed by a statistician at the RCB (AMcl) and verified by a statistician at the BHF Glasgow Cardiovascular Research Centre (AT) after the database was locked according to a pre-specified statistical analysis plan. The RHC cohort required assessment at a single time point and we estimated a sample size of 20 patients would be recruited, giving 80% power to detect a correlation of 0.6 at 5% statistical significance. The hospitalized HF cohort and dialysis cohort required multiple assessments per patient, so we aimed to recruit 40 patients into each cohort, giving protection against missing outcome data, while ensuring at least the same level of statistical power as for the RHC cohort. The study was delayed and interrupted by the COVID-19 pandemic and the total number of patients recruited into the inpatient and dialysis cohort was revised to 20 to match the RHC which preserved power. Continuous variables are summarized using the mean and standard deviation (SD) or median with interquartile ranges (IQR). Categorical variables are summarized with frequencies and percentages. Patient-individual Spearman correlations (r_{sp}) were used to determine the correlations between device measurements and clinical parameters that were continuous variables. Scatter plots graphically report the correlations and heat maps are used to depict multiple correlations where darker colour indicates correlations closer to +1 and lighter colour correlation closer to −1. Relationships between continuous and categorical variables were analysed using Mann–Whitney–Wilcoxon tests or Kruskal–Wallis tests as appropriate. Wilcoxon signed rank and McNemar's tests were used to analyse the relationship between change in continuous or categorical variables, respectively, on serial assessments. A p-value <0.05 was considered statistically significant. Analyses were performed using R (R Foundation for Statistical Computing, Vienna, Austria) and Stata version 18 (College Station, TX, USA).

Safety monitoring

Patients were reviewed within 48 h of a device reading to assess for device-related adverse effects. Patients with pre-existing cardiac

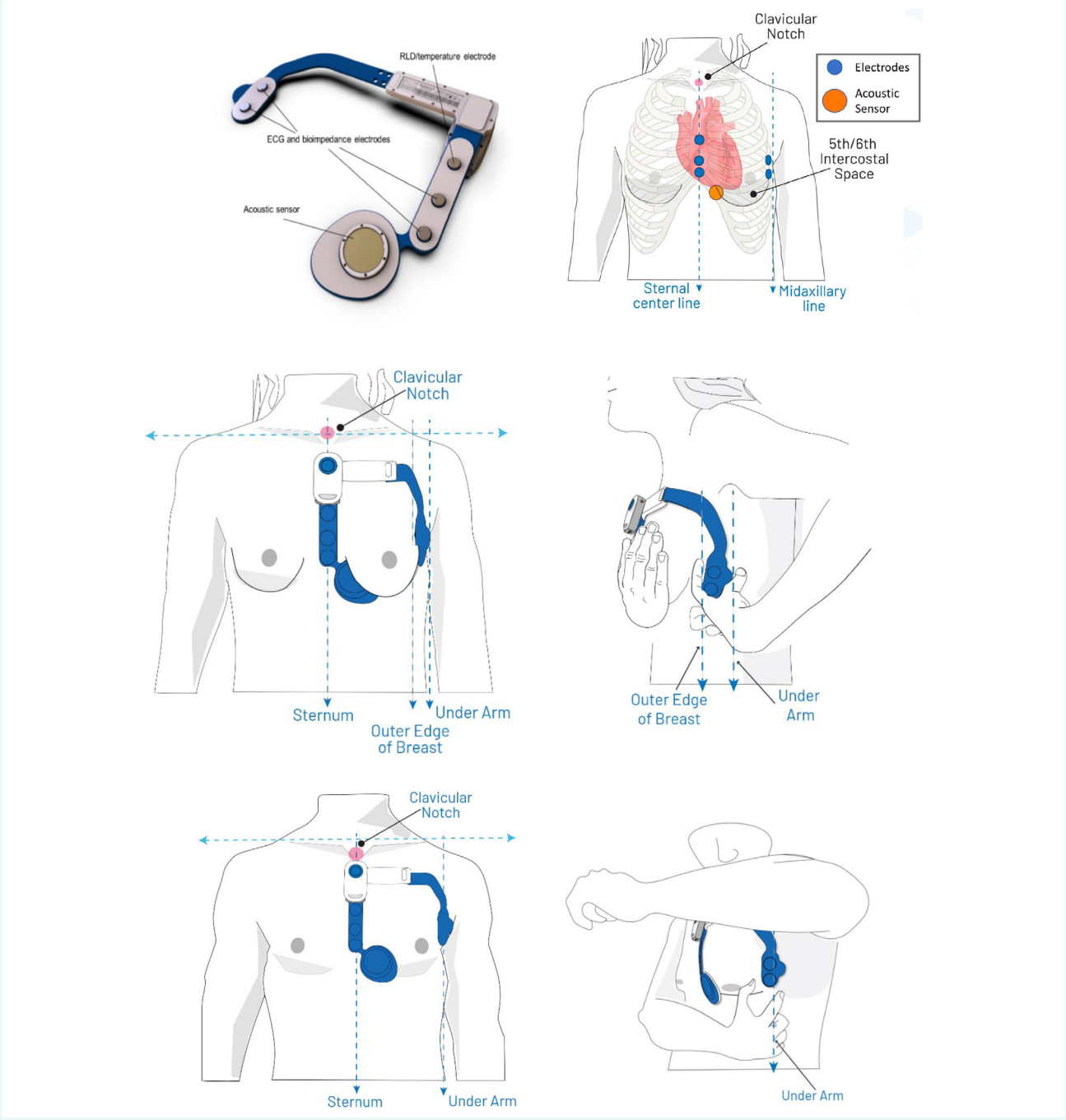


Figure 1 Picture of the device and schematic showing the position of the sensors and underlying anatomical landmarks. Schematic of the location of the device on the left chest on both female and male patients with lateral view of the device placement.

implantable electronic devices (CIED) had real-time monitoring of the CIED while wearable device readings were performed for surveillance of potential device–device interactions.

Ethics and consent

Ethical approval was granted by the London-Dulwich Research Ethics Committee. All patients provided written informed consent prior to enrolment. The study was registered (ClinicalTrials.gov identifier NCT05026034).

Study funding

CONGEST-HF was sponsored by NHS Greater Glasgow and Clyde Research and Innovations. ADI provided funding to the University of Glasgow for the design and conduct of the study but played no role in

the collection of clinical data, the statistical analyses or the decision to publish the results.

Results

Performance of the device in patients hospitalized with heart failure

A total of 25 patients with a primary diagnosis of HF on admission to hospital were recruited. The median time to switch from IV to oral diuretics was 4 days (IQR 3–5) and the median time from enrolment to discharge was 7 days (IQR 4–14). Patients enrolled were representative of hospitalized patients with HF with a mean age of 72 (SD 12) years, mean ejection fraction of 31% (SD 15%), 72% in New York Heart Association (NYHA) class III or IV and NT-proBNP was elevated at 3899 pg/ml (IQR 1567-9151) (*Table 1*). The mean change in weight from day 1 on IV diuretics to the day of discharge was -3.7 ± 5.5 kg ($p < 0.001$) and the number of B-lines on LUS fell from a median of 80 (IQR 49–124) on day 1 of IV diuretics to 44 (IQR 24–59) on day of discharge (p-value for difference with visit 1 < 0.001).

Change in CPM measured thoracic impedance and change in weight between day 1 on IV diuretics and the day of discharge were significantly correlated ($r_{sp} = -0.64$, $p = 0.002$) (*Figure 2*). Correlation of the change in device S3 and change in weight from visit 1 to day of discharge was statistically significant ($r_{sp} = -0.53$, $p = 0.014$) (*Figure 2* and online supplementary *Figure S2*). The correlations between thoracic impedance and S3 with LUS B-lines at each visit or between visits did not reach statistical significance (online supplementary *Tables S2*, *S3* and *Figures S3–S6*).

The CPM device tidal volume correlated with spirometry tidal volume (online supplementary *Table S4*) most clearly on the first visit (online supplementary *Figures S3–S6*). Median values (IQR) are reported for the measured echocardiographic parameters per study visit in online supplementary *Table S5* and correlations (online supplementary *Figures S3–S6* and *Tables S6*, *S7*).

The proportion of patients with symptoms and the ECCS are shown in online supplementary *Table S8* at each visit. Change in total ECCS was correlated with change in device thoracic impedance between day of first IV diuretics and day of discharge ($r_{sp} = -0.44$, $p = 0.045$) (online supplementary *Figure S7*) but not tidal volume or S3 (online supplementary *Figure S2*).

Performance of the device in patients undergoing haemodialysis

A total of 21 patients with end-stage renal disease who were established on intermittent haemodialysis for a minimum of 90 days were enrolled. This cohort was predominantly male with a mean age 60 ± 14 years (*Table 1*). Of the 21, 7 (33%) patients had a history of HF. The majority of people had at least one cardiovascular risk factor, most often hypertension (71.4%) followed by diabetes (28.6%) and a prior myocardial infarction (19.1%). The median volume of fluid removed by haemodialysis was 1999 ml (IQR 1600–2000). The median number of B-lines before dialysis was 58 (33–94) and after dialysis was 31 (15–67) (p-value for difference < 0.001).

The CPM device thoracic impedance and B-lines on LUS were strongly correlated before ($r_{sp} = -0.71$, $p < 0.001$) and after ($r_{sp} = -0.77$, $p < 0.001$) dialysis (online supplementary *Figures S8* and *S9*). The correlation between the change in device thoracic impedance and change in B-lines was weaker and did not reach statistical significance ($r_{sp} = -0.19$, $p = 0.396$) (*Figure 3* and online supplementary *Figure S10*). The correlation between the volume of fluid removed by dialysis and the change in device thoracic impedance was $r_{sp} = 0.49$, $p = 0.024$ (*Figure 3*).

The CPM device S3 was significantly correlated with LUS B-lines before dialysis ($r_{sp} = 0.48$, $p = 0.028$) (online supplementary *Figure S8*). The strength of the correlation was weaker after dialysis ($r_{sp} = 0.40$, $p = 0.075$) (online supplementary *Figure S9*) but the correlation between the change in both parameters was evident but not statistically significant ($r_{sp} = 0.43$, $p = 0.055$) (online supplementary *Figure S10*). Change in CPM S3 ($r_{sp} = -0.24$, $p = 0.283$) and change in CPM tidal volume ($r_{sp} = 0.06$, $p = 0.810$) did not correlate significantly with fluid removed by dialysis (online supplementary *Figure S10*).

There was a correlation between spirometry tidal volume and volume of fluid removed by dialysis that did not reach statistical significance ($r_{sp} = 0.43$, $p = 0.052$) with similar correlations in tidal volume by spirometry and CPM device tidal volume before ($r_{sp} = 0.41$, $p = 0.063$) or after dialysis ($r_{sp} = 0.40$, $p = 0.073$). Change in device tidal volume did not correlate significantly with change in spirometry tidal volume ($r_{sp} = 0.28$, $p = 0.219$). Echocardiography was performed and median values (IQR) are reported for the measured echocardiographic parameters per study visit in online supplementary *Table S9* and correlations between CPM device thoracic impedance, S3 and tidal volume and echocardiography measures before and after dialysis (online supplementary *Tables S10*, *S11* and *Figures S8*, *S9*) were inconsistent.

Change in total ECCS was correlated significantly with CPM device thoracic impedance ($r_{sp} = -0.44$, $p = 0.046$) (online supplementary *Figure S11*).

Performance of the device in patients undergoing right heart catheterization

The characteristics of the patients enrolled are shown in *Table 1* and were representative of a population with advanced HF. Device-measured S3 and PCWP obtained at a single time-point were not significantly correlated ($r_{sp} = 0.30$, $p = 0.204$) (*Table 2* and online supplementary *Figure S12*). On analysis of additional RHC measures, pulmonary vascular resistance on RHC was significantly correlated with device thoracic impedance ($r_{sp} = -0.47$, $p = 0.036$). Cardiac index (calculated using the Fick equation) was inversely correlated with tidal volume measured by the CPM device ($r_{sp} = -0.47$, $p = 0.043$) (*Table 2*).

The CPM device tidal volume and spirometry tidal volume were significantly correlated ($r_{sp} = 0.45$, $p = 0.049$). B-lines on LUS were significantly correlated with CPM S3 ($r_{sp} = 0.45$, $p = 0.046$), CPM thoracic impedance ($r_{sp} = -0.57$, $p = 0.010$) and CPM tidal volume ($r_{sp} = 0.47$, $p = 0.036$). There were several correlations between measures on echocardiography and CPM device measures (online supplementary *Table S12*, *Figure S12*).

Table 1 Baseline characteristics and therapy for patients enrolled in each cohort

	Hospitalized patients with HF ($n = 25$)	Dialysis cohort ($n = 21$)	RHC cohort ($n = 20$)
Male sex, *n* (%)	18 (72)	15 (71)	14 (70)
Age (years)	72 ± 12	60 ± 14	55 ± 9
Race, *n* (%)			
White	23 (92)	19 (91)	20 (100)
Asian	2 (8)	2 (9)	-
BMI (kg/m^2)	29 ± 5	25 ± 5	27 ± 4
Medical history, *n* (%)			
Hypertension	12 (48)	15 (71)	1 (5)
Diabetes	7 (28)	6 (29)	3 (15)
AF on baseline ECG	7 (30)	5 (24)	4 (20)
History of any AF	12 (48)	6 (29)	9 (45)
MI	7 (28)	4 (19)	4 (20)
Stroke	4 (16.0)	2 (10)	1 (5)
COPD	3 (12)	1 (5)	1 (5)
LVEF (%)	31 ± 15	48 ± 17	30 ± 11
NYHA class, *n* (%)			
I	0 (0)	4 (19)	0 (0)
II	7 (28)	5 (24)	7 (35)
III	10 (40)	9 (43)	12 (60)
IV	8 (32)	3 (14)	1 (5)
Symptoms/signs, *n* (%)			
Dyspnoea	9 (36)	16 (76)	19 (95)
Orthopnoea	13 (52)	8 (38)	9 (45)
PND	4 (16)	5 (24)	3 (15)
Fatigue	21 (84)	19 (91)	20 (100)
Bendopnoea	11 (44)	9 (43)	13 (65)
Peripheral oedema	22 (88)	6 (29)	4 (20)
Systolic BP (mmHg)	121 ± 25	138 ± 34	103 ± 13
Heart rate (bpm)	79 ± 17	80 ± 15	75 ± 19
Respiratory rate (/min)	17 ± 2	16 ± 1	16 ± 2
Chest circumference (cm)	108 ± 13	102 ± 13	108 ± 10
Duration of HF, *n* (%)			
<1 year	9 (36)	3 (14)	3 (15)
1–5 years	13 (52)	3 (14)	7 (35)
>5 years	3 (12)	1 (5)	10 (50)
Ischaemic aetiology, *n* (%)	11 (44)	3 (14)	6 (30)
Prior HF hospitalization, *n* (%)	13 (52)	1 (5)	11 (55)
HF hospitalization within previous 6 months, *n* (%)	6 (24)	1 (5)	3 (15)
NT-proBNP (pg/ml)	3899 (1567–9151)	43 624 (4955–88 720)	2116 (784–4341)
eGFR (ml/min/1.73 m^2)	49 (33–69)	7 (5–10)	59 (45–77)
Baseline treatment, *n* (%)			
Intravenous diuretics	25 (100)	5 (24)	1 (5)
Loop diuretic	0 (0)	0 (0)	18 (90)
Thiazide/thiazide-like diuretic	8 (32)	5 (24)	1 (5)
ACE inhibitor or ARB	6 (24)	1 (5)	4 (20)
Sacubitril/valsartan	17 (68)	16 (76)	12 (60)
Beta-blocker	9 (36)	0 (0)	17 (85)
MRA	11 (44)	0 (0)	15 (75)
SGLT2 inhibitor	1 (4)	1 (5)	13 (65)
Digoxin	4 (16)	1 (5)	2 (10)
Any implanted device	2 (8)	0 (0)	12 (60)

Table 1 (Continued)

	Hospitalized patients with HF ($n = 25$)	Dialysis cohort ($n = 21$)	RHC cohort ($n = 20$)
ICD	0 (0)	0 (0)	11 (55)
CRT-D	0 (0)	0 (0)	1 (5)
CRT-P	2 (8)	1 (5)	0 (0)
Pacemaker	0 (0)	0 (0)	0 (0)

ACE, angiotensin-converting enzyme; AF, atrial fibrillation; ARB, angiotensin receptor blocker; BMI, body mass index; BP, blood pressure; COPD, chronic obstructive pulmonary disease; CRT-D, cardiac resynchronization therapy-defibrillator; CRT-P, cardiac resynchronization therapy-pacemaker; ECG, electrocardiogram; eGFR, estimated glomerular filtration rate; HF, heart failure; ICD, implantable cardioverter-defibrillator; LVEF, left ventricular ejection fraction; MI, myocardial infarction; MRA, mineralocorticoid receptor antagonist; NT-proBNP, N-terminal pro-B type natriuretic peptide; NYHA, New York Heart Association; PND, paroxysmal nocturnal dyspnoea; RHC, right heart catheterization; SGLT2, sodium–glucose cotransporter 2.

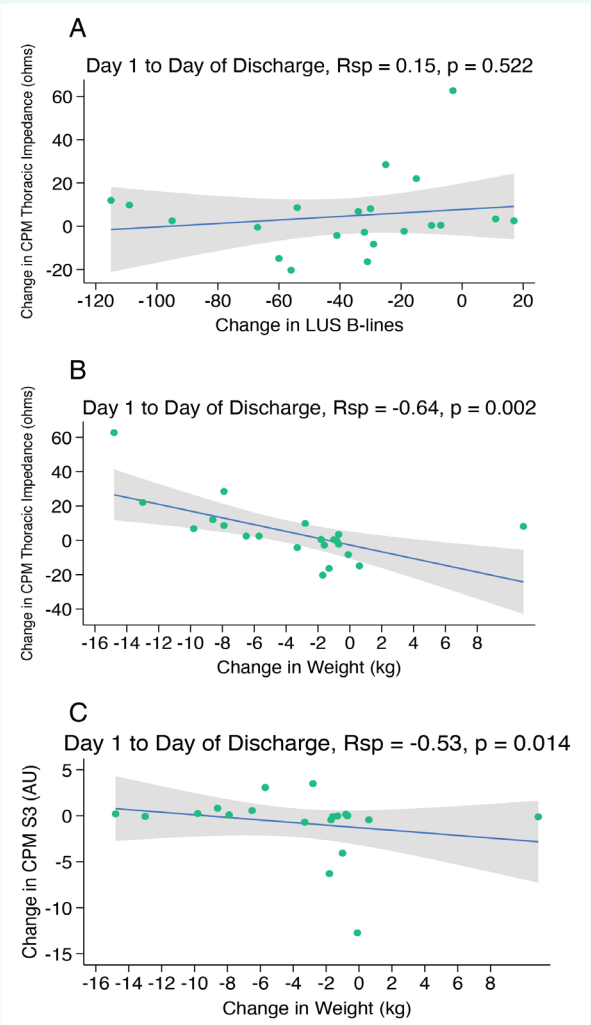


Figure 2 Correlation between change in device-measured thoracic impedance and number of B-lines on lung ultrasound (LUS) (*A*), and change in body weight (*B*) and correlation between change in device-measured third heart sound (S3) and change in body weight (*C*). CPM, cardiopulmonary monitoring; Rsp, Spearman correlation.

There was a trend of increasing values in CPM device S3, thoracic impedance and tidal volume across higher grades of the EVEREST dyspnoea scale that did not reach statistical significance (online supplementary *Table S13*). A similar general trend was observed across NYHA functional classes (online supplementary *Table S13*).

Adverse events and device deficiencies

There were no device-related adverse events in this study cohort, including no evidence of device–device interaction in any patients with an implanted cardiac device. Six device deficiencies occurred, two were due to the adhesive falling off and required replacement, three deficiencies were due to the device being unable to obtain an auscultation wave form, and one case where the device failed to connect to the mobile application.

Discussion

In this study, the change in thoracic impedance measured by the non-invasive monitoring device correlated with change in weight though not B-lines on LUS in patients hospitalized for HF as their congestion was treated, and was also correlated with patient symptoms as measured by the ECCS. In patients receiving haemodialysis change in thoracic impedance was correlated with volume of fluid removed during the dialysis session (*Graphical Abstract*).

We examined the correlation between multiple measures derived from the CPM device and clinical measures related to congestion at single time points and with changes in the measures. We found that correlations were most pronounced with changes in keeping with the purpose of the device, to detect changes in a patient over time. Correlations between CPM-derived measures and clinical measures that were not statistically significant were in a direction that was in keeping with a plausible physiological relationship. The correlation with a single measurement at RHC was low and this was in part expected. The device is designed to track changes within a patient rather than changes or differences between patients and static measures. The most robust correlations were observed in the patients undergoing decongestion during a hospitalization for HF, the target population.

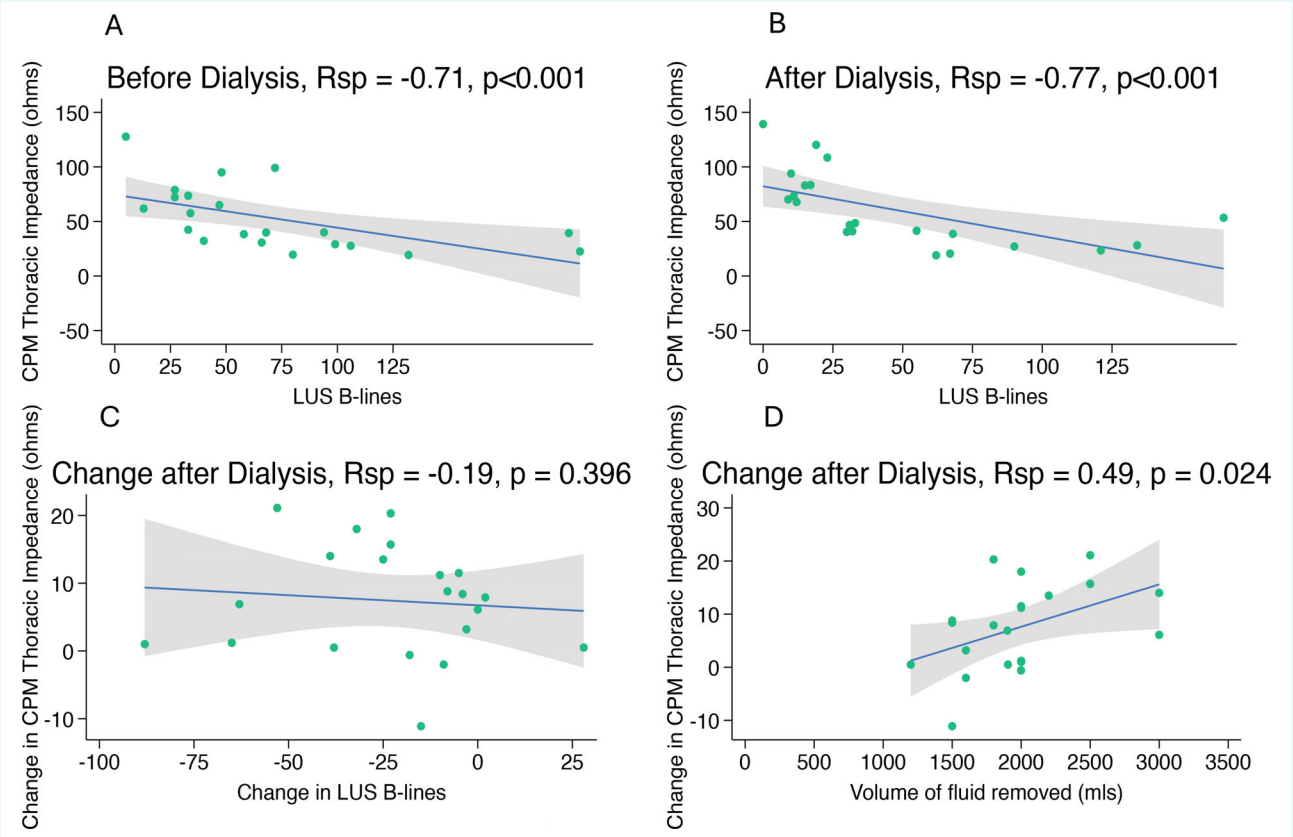


Figure 3 Correlation between change in device-measured thoracic impedance and number of B-lines on lung ultrasound (LUS) before (*A*) and after (*B*) dialysis and change in thoracic impedance and change in B-lines on LUS (*C*) and change in thoracic impedance and volume of fluid removed during dialysis (*D*). CPM, cardiopulmonary monitoring; Rsp, Spearman correlation.

Table 2 Correlation between device-derived third heart sound, thoracic impedance and tidal volume and measures from the right heart catheterization

RHC parameter	CPM device S3		CPM device thoracic impedance		CPM device tidal volume	
	r_{sp}	*p*-value	r_{sp}	*p*-value	r_{sp}	*p*-value
Cardiac output (thermodilution)	−0.23	0.338	0.29	0.211	−0.30	0.204
Cardiac index (thermodilution)	−0.27	0.250	0.25	0.285	−0.24	0.309
Cardiac output (Fick)	−0.29	0.227	0.32	0.185	−0.42	0.068
Cardiac index (Fick)	−0.40	0.093	0.36	0.135	−0.47	0.043
PA pressure – mean	0.29	0.208	−0.21	0.364	0.30	0.195
PA pressure – systolic	0.25	0.288	−0.07	0.755	0.22	0.355
PA pressure – diastolic	0.35	0.130	−0.22	0.343	0.36	0.117
PA saturation	−0.29	0.225	0.33	0.167	−0.33	0.164
RA pressure	0.04	0.863	0.16	0.499	−0.12	0.610
Pulmonary vascular resistance	0.25	0.279	−0.47	0.036	0.40	0.083
Systemic vascular resistance	0.31	0.176	−0.25	0.289	0.29	0.214
PCWP	0.30	0.204	−0.08	0.729	0.27	0.248

CPM, cardiopulmonary monitoring; PA, pulmonary artery; PCWP, pulmonary capillary wedge pressure; RA, right atrial; RHC, right heart catheterization; r_{sp}, Spearman correlation; S3, third heart sound.

We were able to observe correlations between changes in fluid status over a short period of time in those patients undergoing haemodialysis. As can be seen from the results, multiple correlations exist between the measures from the CPM device and the clinical measures, and by integrating the information from each of the measurements taken, the sensitivity of the device to detect subclinical decompensation could be improved and indicate that a multiparameter approach to monitoring HF is useful.

We studied correlations with clinically recognized and meaningful measures of congestion. It may be surprising that in our cohort of patients hospitalized for HF, thoracic impedance did not correlate well with LUS B-lines, a sensitive marker of pulmonary fluid and predictor of rehospitalization in HF.[9,10] However, this may be because all patients were congested on admission at the start of the study, and many remained congested as indicated by the presence of B-lines at the time they were determined fit for discharge by the treating team. Therefore, the difference in B-lines may not have been large enough to detect a statistically significant correlation and perhaps with greater decongestion, the correlation would be evident. A further issue may be the phenotype of decompensated patients with HF. Patients can present with predominantly pulmonary signs but can also present with weight gain and peripheral oedema.[11–13] Given that correlations between changes in CPM-derived thoracic impedance and S3 correlated with weight change, this would suggest these indices were a more sensitive measure of decongestion in this population who perhaps had more peripheral than pulmonary congestion.

The device is designed to be used by one patient over time to monitor HF-related health status and warn of possible decompensation. Our findings suggest that the device can detect physiological changes associated with the worsening of HF and that it may be possible to develop an algorithm to provide early warning of decompensation and allow appropriate intervention. Clinical examination in HF is insensitive and dependent on experience, especially for specific signs such as a S3.[14] The ability of the CPM device to detect S3 energy, in essence an inaudible diastolic heart sound, and its correlation with clinically meaningful changes in congestion, like change in body weight, suggest that the device could help aid decongestion by identifying otherwise difficult to detect signs and integrating these into the assessment of the patient.

Our study has several limitations. We enrolled a relatively small number of patients, although our studies were powered to make clinically meaningful correlations between the measures derived from the device and clinically relevant markers of congestion and decongestion. We did not test the device in the outpatient setting where the device was designed to detect congestion and decompensation. We did not test whether using this device could reduce hospitalizations and improve outcomes and this remains to be tested in a randomized trial. In many patients the symptoms of HF are only evident on exertion and we obtained all measurements at rest. Whether sensitivity could be improved with measurement during or shortly after exercise is to be determined in an ongoing study.

Conclusion

A non-invasive monitoring device worn for less than 5 min was able to measure changes in congestion in patients undergoing decongestion in hospital for decompensated HF and fluid changes in patients requiring haemodialysis. The device, measuring multiple parameters, provides a new method of monitoring patients with HF.

Supplementary Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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