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Wearable vest for pulmonary congestion tracking and prognosis in heart failure: A pilot study

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Given the epidemic of readmissions for heart failure (HF) in western countries, new tools for congestion tracking are needed in the management of patients with HF. We previously presented a novel, wearable textile vest that could track decongestion during therapy for acutely decompensated HF (ADHF) in a cardiac ward. The vest wraps around the chest and has textile electrodes placed on the lateral sides so that multi-frequency measurements of bio-impedance can be taken of the whole chest. The vest data showed strong agreement with fluid loss as determined by weight changes and good correlation with changes in signs and symptom (HF severity score) [1].

Here, we assessed the ability of this vest to provide additional insight into the prognosis of these patients. We included 20 patients admitted to the cardiology ward for ADHF, and we followed them for 18 months. No patient was lost to follow-up. Inclusion/exclusion criteria and in-hospital measurements and their correlations have been described elsewhere [1]. Upon discharge, patients were clinically followed-up at 3 months and 12 months. The vital status was determined at 18 months post-discharge via medical records. Clinical assessments involved a physical examination, which included an assessment of the New York Heart Association (NYHA) functional class, a clinician-assessed HF severity score (HFSS), based on the Framingham congestion criteria [2] to assess HF decompensation [3,4], and a measure of body weight. Clinical assessments also included an ECG, blood work, echocardiography, and bioimpedance, measured with the novel vest (measured in a semi-recumbent position with the couch lifted to 30° for 5 min). Changes in extracellular fluid levels were estimated by R_0. Descriptive analyses were performed at the first step. Categorical variables were described by frequencies and percentages. Continuous variables were described by means and standard deviations after having assessed normal distribution by normal Q–Q plots. Baseline statistical differences between groups were assessed using the chi-square test for categorical variables, Student's t-test for continuous variables. Statistical analyses were performed using the SPSS 11 statistical package (SPSS Inc., Chicago, IL). A 2-sided $p < 0.05$ was considered significant. The study was approved by the Hospital Ethics Committee.

During the follow-up, 6 patients died (30%), 4 due to pump failure (20%), 1 due to acute myocardial infarction and 1 committed suicide. Patient characteristics are shown in Table 1. We also compared characteristics between patients that died from pump failure and patients that did not died from HF (alive patients or dead from other causes, Table 1).

Fig. 1 shows impedance values (R_0) during hospital admission and follow-up. A threshold value below 20 Ω was identified post-hoc in a discretionary way based on the obtained graph as prognostically meaningful (Fig. 1). At hospital admission, nine patients had low impedance values ($R_0 < 20$ Ω). Impedance remained low (below this threshold) in four of these patients at discharge; moreover, three of these patients (75%) died of pump failure during the follow-up period.

At discharge, the impedance in 16 patients had raised to ≥20 Ω (irrespective of admission value); of these, 15 were alive for the entire follow-up period; two patients dropped below 20 Ω at the 12-month follow-up, but remained alive at 18 months of follow-up (Fig. 1); and one patient, whose impedance improved considerably during the hospital admission (discharge $R_0 = 33$ Ω), experienced a drop to <20 Ω at 3-months, and finally ended in pump failure and death. The R_0 values at discharge were significantly different between patients who died from HF and others ($p = 0.02$).

It was noteworthy that the percentage of improvement was not as important for survival as achieving an R_0 value >20 Ω. In point of fact, the patients who died had achieved greater in-hospital improvements

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Table 1
Demographic, clinical, biochemical, and pharmacological treatment data for the study population, grouped by vital status at 18 months.

Characteristics	Total cohort N = 20	18 month follow-up		
		Did not die from HF N = 16	Died from HF N = 4	p-Value
Age (years)	74.7 ± 9.5	73.8 ± 10.5	78.3 ± 1.7	0.41
Women (%)	3 (15%)	3 (15%)	0	0.49
Etiology (%)				0.31
Ischemic heart disease	9 (45%)	6 (37.5%)	3 (75%)	
Dilated cardiomyopathy	3 (15%)	2 (12.5%)	1 (25%)	
Toxic cardiomyopathy	1 (5%)	1 (5%)	0	
Valvular disease	1 (5%)	1 (5%)	0	
Other	6 (30%)	6 (30%)	0	
Heart failure duration (years)	2.6 ± 4.1	1.9 ± 3.1	5.4 ± 6.7	0.12
LVEF (%)	37.0 ± 12.5	38.4 ± 11.7	31.3 ± 15.5	0.32
HR (bpm)	87.0 ± 16.4	88.1 ± 17.4	82.5 ± 12.4	0.56
SBP (mm Hg)	126.8 ± 20.6	128.3 ± 18.5	120.8 ± 30.4	0.53
DBP (mm Hg)	73.7 ± 10.0	74.0 ± 9.5	72.5 ± 13.5	0.8
NTproBNP (pg/mL)	6761 ± 6672	4724 ± 5122	14,905 ± 6338	0.003
BMI (kg/m^2)	26.1 ± 4.5	27.3 ± 4.3	21.6 ± 0.7	0.02
Fat mass (%)	27.2 ± 6.4	28.7 ± 5.7	21.3 ± 5.7	0.03
R_0 at admission (Ω)	21.8 ± 7.7	24.1 ± 6.6	12.4 ± 2.7	0.003
Comorbidities (%)				
Hypertension	17 (85%)	13 (81.3%)	4 (100%)	0.49
Diabetes mellitus	8 (40%)	6 (37.5%)	2 (50%)	0.54
COPD	4 (20%)	3 (18.8%)	1 (25%)	0.62
eGFR < 60 mL/min/1.73 m^2	7 (35%)	4 (25%)	3 (75%)	0.1
Atrial fibrillation	9 (45%)	8 (50%)	1 (25%)	0.45
Treatment (%)				
ACEI/ARB	11 (55%)	11 (68.8%)	0	0.03
Beta-blockers	12 (60%)	9 (56.3%)	3 (75%)	0.47
Loop diuretics	20 (100%)	15 (93.8%)	3 (75%)	0.37
MRAs	6 (30%)	5 (31.3%)	1 (25%)	0.66
Digoxin	10 (50%)	6 (37.5%)	2 (50%)	0.53

Data expressed as mean ± standard deviation, or absolute number (percentage).
ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; BMI, body mass index; COPD, chronic obstructive pulmonary disease; CrCl, creatinine clearance; DBP, diastolic blood pressure; HR, heart rate; LVEF, left ventricular ejection fraction; SBP, systolic blood pressure; MRA, mineralocorticoid receptor antagonist.

than alive patients (Δ% of improvement in mean R_0: 39.2 ± 35.2% for alive patients vs. 63.2 ± 64.8% for patients who died, p = 0.32).

NTproBNP, a marker of myocyte stretch and congestion, showed a good correlation with R_0, both at admission for acute decompensation (R = −0.57, p = 0.009) and at the ambulatory 1 year follow-up visit (R = −0.59, p = 0.04).

This promising data suggested that a portable vest that non-invasively tracks impedance may provide a refinement in the prognostic assessment of patients admitted for HF beyond the clinical improvement. Wearable devices hold potential for reducing HF readmissions and costs. However, future studies are needed with larger cohorts to gain stronger evidence to support the translation of this approach into clinical practice.

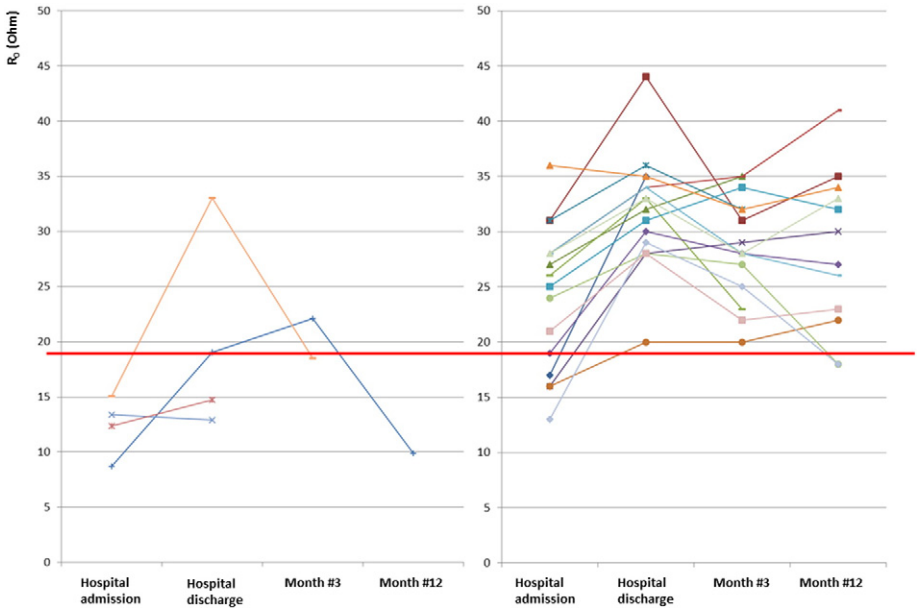

Fig. 1. R_0 values during hospital admission and follow-up for patients that died of pump failure (left panel) compared to the rest of the patient cohort during follow-up (right panel). Red line divides the points below and above 20 Ω.

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Disclosures

I. Cuba-Gyllensten is a PhD student at Philips Research, and J. Riistama and R. Aarts are researchers at Philips Research.

Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.ijcard.2016.04.024.

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