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ARTICLE

SMART-HF: structured management approach to remote treatment of heart failure. Guiding best practices

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Abstract

Responses to remote pulmonary artery pressure data vary across providers, days, and institutions, and only a minority of patients with elevated pressures reach targets linked to survival under *ad hoc* management. We evaluated SMART-HF, a structured pulmonary artery diastolic pressure (PAD)-guided workflow, in a community heart failure cohort. We retrospectively analyzed 37 adults (mean age 75.1±10.4 years; 17 [45.9%] female; left ventricular ejection fraction ≥50% in 23 [62.2%]; atrial fibrillation or flutter in 24 [64.9%]; hypertension in 32 [86.5%]; mean eGFR 54.9±21.3 mL/min/1.73 m^2; median NT-proBNP 1846 [IQR 813-4036] pg/mL) with an implanted pulmonary artery pressure sensor managed with SMART-HF. PAD was averaged over prespecified 14-day windows at baseline, 90 days, and 6 months. Two prespecified hemodynamic management performance indices (HMPI) were defined: the 90-Day Target HMPI (PAD ≤20 mmHg at 90 days) and the 6-Month Delta HMPI (PAD reduction >2 mmHg from baseline). Thirty-six patients had paired 90-day and 29 had paired 6-month windows. Mean PAD decreased from 18.3±7.0 to 16.1±6.3 mmHg at 90 days (change −2.1±3.3 mmHg; 95% CI −3.2 to −1.0; P<0.001) and from 18.8 ± 6.8 to 15.5 ± 5.8 mmHg at 6 months (change −3.3±4.3 mmHg; 95% CI −4.9 to −1.7; P<0.001). The 90-Day Target HMPI was met in 26/36 (72.2%; 95% CI 54.8–85.8; P=0.011) and the 6-Month Delta HMPI in 19/29 (65.5%; 95% CI 45.7–82.1). Nineteen patients (51.4%) had baseline PAD >20 mmHg; in this subgroup PAD fell by −2.9±3.6 mmHg at 90 days (n=19; P=0.002) and −4.9±4.9 mmHg at 6 months (n=15; P=0.002), with the 6-Month Delta HMPI met in 12/15 (80.0%; P=0.035) – approximately double the 24% target-attainment rate reported for comparable patients under *ad hoc* management. No serious adverse events or heart-failure hospitalizations occurred. Seven patients required management for electrolyte derangements and eight met KDIGO criteria for acute kidney injury during decongestion; in five, transmitted pressure waveforms revealed occult paroxysmal atrial fibrillation, and after successful restoration of sinus rhythm, all five continued to decongest safely. SMART-HF was associated with improved ambulatory pulmonary artery pressures in patients with either elevated or controlled baseline averages and a reassuring safety profile supportive of additional large-scale research.

Key words: heart failure; pulmonary artery pressure; CardioMEMS; remote monitoring; congestion; implementation.

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Introduction

The growing burden of heart failure (HF) in the United States continues to outpace multidisciplinary efforts to reduce morbidity and mortality. While 5-year survival for many malignancies now approaches 70%,[1] those afflicted with HF are faced with persistently poor outcomes; 5-year survival after an index HF hospitalization has been reported near 25% regardless of left ventricular ejection fraction.[2]

Guideline-directed medical therapies (GDMT), close follow-up, and implantable pulmonary artery (PA) pressure monitoring have been established as effective tools to reduce congestion-related HF events. However, real-world adoption and execution of these proven interventions remain heterogeneous, with management differing materially between providers and systems. Even when real-time hemodynamic data are available, treatment decisions may still rely on delayed clinical surrogates (symptoms, weight changes, and ex-

amination findings), delaying recognition and treatment of congestion.

Evidence linking ambulatory PA pressures to outcomes is no longer theoretical. Large, pooled analyses have demonstrated that ambulatory PA pressures and their change over time independently predict subsequent mortality.[3] Complementary evidence from real-world datasets suggests that under *ad hoc* management, a minority of patients with elevated baseline PAD achieve PAD thresholds associated with improved survival,[3,4] highlighting an implementation gap that may be addressable with standardized, reproducible workflows.

We propose SMART-HF (Structured Management Approach to Remote Treatment of Heart Failure), a PAD-centric, algorithm-driven workflow designed to reduce variability in response to remote PA pressure data and systematically drive PAD towards physiologically normal targets. The primary aim of the present study was to evaluate the hemodynamic effectiveness of SMART-HF by quantifying change in PAD and PAD target attainment using prespecified 14-day averaged windows.

Materials and Methods

Study design and setting

Single-center retrospective cohort analysis of adults with heart failure implanted with a remote pulmonary artery pressure sensor and managed exclusively using the SMART-HF workflow at Froedtert Menomonee Falls Hospital (Menomonee Falls, Wisconsin, USA). Implant dates in the analyzed cohort ranged from 20 September 2024 to 11 December 2025.

Participants

Eligibility followed current United States Food and Drug Administration indications for the CardioMEMS HF System. Under original labelling, patients with New York Heart Association (NYHA) class III heart failure and a heart failure hospitalization within the preceding 12 months were eligible. Under expanded indications, patients with NYHA class II heart failure without a recent heart failure hospitalization were eligible if N-terminal pro-B-type natriuretic peptide (NT-proBNP) met body mass index–indexed thresholds stratified by left ventricular ejection fraction category. All adults (≥18 years) with an implanted remote pulmonary artery pressure sensor at Froedtert Menomonee Falls Hospital who were managed exclusively via SMART-HF were included. No additional exclusion criteria were applied beyond standard contraindications to pulmonary artery pressure sensor implantation.

Sex was ascertained from the electronic health record and reported as a baseline characteristic (Table 1). Both male and female patients were included without restriction. The modest sample size (n=37) precluded prespecified sex-stratified analyses of hemodynamic outcomes; however, sex was examined as a descriptive covariate.

SMART-HF workflow

Operationalized review of the entire cohort was performed in accordance with previously published methodology.[5] Remote pulmonary artery pressures were reviewed by advanced practice providers every Monday, Wednesday, and Friday. Patient-specific pulmonary artery diastolic pressure targets were set at time of sensor implantation. For patients with right heart catheterization PAD values of 25 mmHg or lower, 15 mmHg was designated as goal pressure. For individuals with significant congestion evidenced by invasive PAD values of >25 mmHg, measured PAD value minus 10 mmHg derived the initial target pressure which was subsequently refined during the early post-implant period (days 5-7) with ideal goal for most patients being 15 mmHg.

Pharmacologic therapy was augmented according to prespecified triggers and thresholds after three serial pulmonary artery diastolic pressure transmissions met criteria relative to the patient's target. Triggers and actions were as follows: pulmonary artery diastolic pressure ≥5 mmHg below target prompted stopping metolazone (if used) and/or decreasing loop diuretic frequency; pulmonary artery diastolic pressure 3-5 mmHg above target prompted increasing loop diuretic frequency to twice daily for two days and/or (if on furosemide) transitioning to an equivalent dose of torsemide or bumetanide; pulmonary artery diastolic pressure ≥6 mmHg above target prompted adding metolazone (renal-adjusted dose) and/or doubling loop diuretic frequency for three days and/or switching to torsemide or bumetanide with an approximately 25% dose increase. Additional safety rails, maximum daily dosing, and laboratory monitoring are summarized in Figure 1.

Hemodynamic data and pulmonary artery diastolic pressure windows

Pulmonary artery diastolic pressure was evaluated according to previously described 14-day windows wherein all available transmitted values within the prespecified window were averaged.[3] Baseline pulmonary artery diastolic pressure was defined as the mean during Days 1-14 post-implant. The 90-day pulmonary artery diastolic pressure window was defined as Days 76-89, and the 6-month window was defined as Days 166-179. Averaged values were included in analysis if they contained ≥5 transmissions at baseline and ≥4 transmissions in the 90-day or 6-month window as defined in prior publications.[3,4] Data were locked for analysis on 25 April 2026; eligibility for the 90-day and 6-month windows was assessed independently, such that a patient with sufficient transmissions for the 90-day window but who had not yet reached or completed the 6-month window contributed only to the 90-day analysis. Subgroup analyses applied the same paired-window inclusion rule within the elevated-baseline subset.

Outcomes

The primary outcome was change in pulmonary artery diastolic pressure from baseline to approximately 6 months using paired

14-day windows. Secondary hemodynamic outcomes included change in pulmonary artery diastolic pressure at 90 days. Two hemodynamic management performance indices (HMPI) were prespecified. The 90-Day Target HMPI was defined as the proportion of patients achieving pulmonary artery diastolic pressure ≤20 mmHg at 90 days; among patients with elevated baseline pressures (>20 mmHg), this transition has been independently associated with lower all-cause mortality.[4] The 6-Month Delta HMPI was defined as the proportion achieving a pulmonary artery diastolic pressure reduction >2 mmHg from baseline to 6 months, a threshold associated with a 14.7% decrease in mortality risk.[3] Exploratory subgroup analyses evaluated the same outcomes and HMPI in patients with baseline pulmonary artery diastolic pressure >20 mmHg (Table 2).

Statistical analysis

Continuous variables are reported as mean ± standard deviation or median (interquartile range), as appropriate. Paired changes in pulmonary artery diastolic pressure were assessed using paired t-tests. Proportions are reported with exact binomial 95% confidence intervals (CI) with corresponding two-sided P-values from the exact binomial test. Statistical software: R version 4.5.3 (R Foundation for Statistical Computing, Vienna, Austria).

Table 1. Baseline characteristics (baseline pulmonary artery diastolic pressure window cohort).

Characteristic	Baseline cohort (n=37)
Demographics	
Age, years	75.1±10.4
Female sex, n (%)	17 (45.9%)
Body mass index, kg/m²	32.6±9.2
First Implant Date, month/day/year	09/20/24
Last Implant Date, month/day/year	12/11/25
Left ventricular ejection fraction classification	
Preserved (LVEF ≥50%), n (%)	23 (62.2)
Mildly reduced (LVEF 41–49%), n (%)	2 (5.4)
Reduced (LVEF ≤40%), n (%)	12 (32.4)
Baseline laboratory values	
eGFR, mL/min/1.73 m²	54.9±21.3
Creatinine, mg/dL	1.39±0.66
NT-proBNP, pg/mL, median [IQR] (n=37)	1846 [813-4036]
Pulmonary artery diastolic pressure	
Mean baseline (14-day average), mmHg	18.2±6.9
Patients with elevated baseline (>20 mmHg), n (%)	19 (51.4)
Comorbid conditions	
Atrial fibrillation/flutter, n (%)	24 (64.9)
Chronic obstructive pulmonary disease, n (%)	11 (29.7)
Coronary artery disease, n (%)	24 (64.9)
Diabetes mellitus, n (%)	10 (27.0)
Home oxygen, n (%)	1 (2.7)
Hypertension, n (%)	32 (86.5)
Guideline-directed medical therapies	
Beta-blocker, n (%)	29 (78.4)
HFpEF (LVEF ≥50%)	18/23 (78.3)
HFmrEF/HFrEF (LVEF <50%)	11/14 (78.6)
ACEi/ARB/ARNI, n (%)	19 (51.4)
HFpEF (LVEF ≥50%)	9/23 (39.1)
HFmrEF/HFrEF (LVEF <50%)	10/14 (71.4)
MRA, n (%)	22 (59.5)
HFpEF (LVEF ≥50%)	14/23 (60.9)
HFmrEF/HFrEF (LVEF <50%)	8/14 (57.1)
SGLT2 inhibitor, n (%)	16 (43.2)
HFpEF (LVEF ≥50%)	10/23 (43.5)
HFmrEF/HFrEF (LVEF <50%)	6/14 (42.9)

ACEi, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor-neprilysin inhibitor; COPD, chronic obstructive pulmonary disease; eGFR, estimated glomerular filtration rate; HFmrEF, heart failure with mildly reduced ejection fraction; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid receptor antagonist; IQR, interquartile range; NT-proBNP, N-terminal pro-B-type natriuretic peptide; PAD, pulmonary artery diastolic pressure; SGLT2, sodium-glucose cotransporter 2.
For medication rows, LVEF <50% denotes the combined mildly reduced/reduced ejection fraction subgroup (n=14).

Results

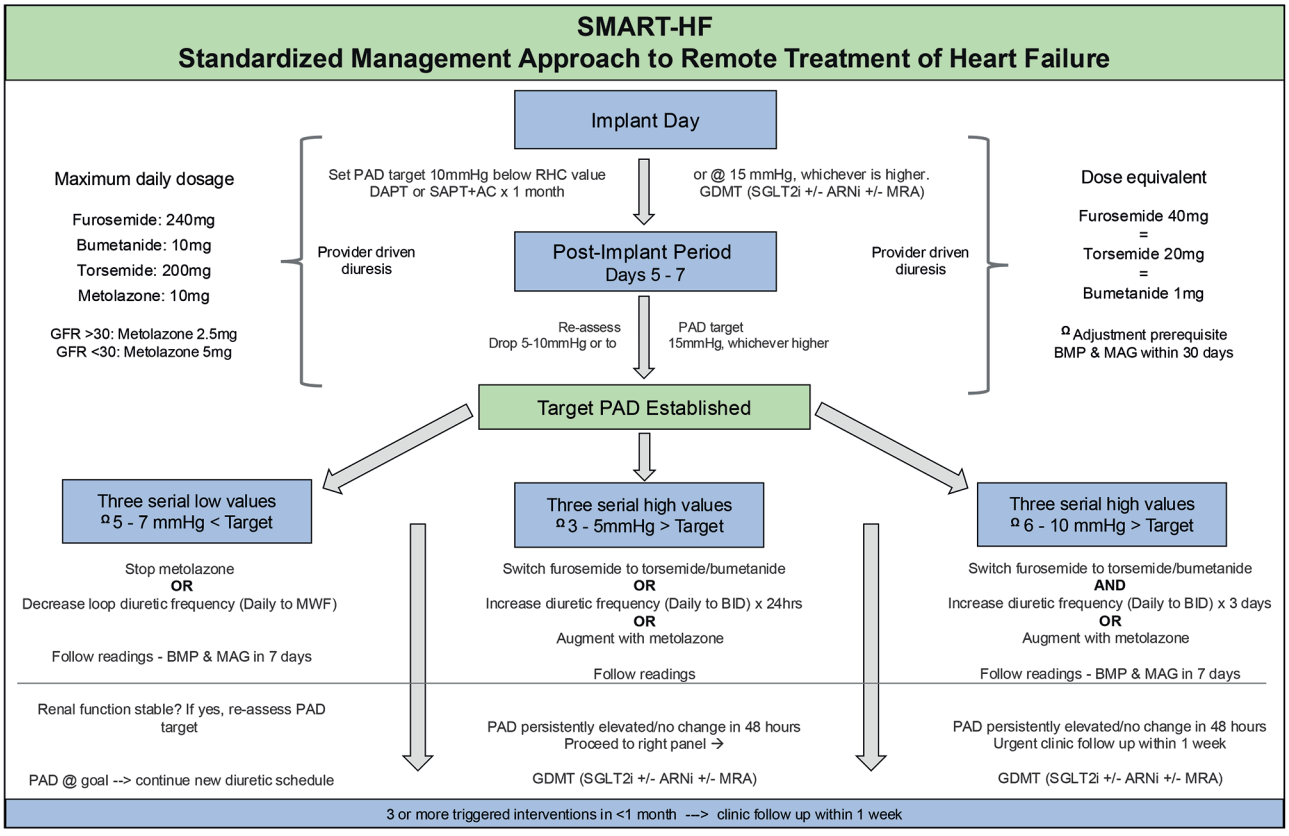
Cohort and analyzable windows

Thirty-seven patients met the baseline transmission threshold (≥5 readings in Days 1-14). Thirty-six had paired baseline and 90-day analyzable windows (≥4 readings in Days 76-89), and 29 had paired baseline and 6-month analyzable windows (≥4 readings in Days 166-179). Eight patients lacked an analyzable 6-month window because these patients had been implanted fewer than six months before data extraction. One patient was excluded from the 90-day analysis because only three transmissions were available in the window. Patient flow through the paired-window analyses is summarized in Figure 2.

Table 2. Hemodynamic outcomes (paired 6-month analysis).

Outcome	Overall paired cohort (n=29)	Baseline PAD >20 subgroup (n=15)
Baseline PAD, mmHg	18.8±6.8	24.3±3.7
6-month PAD, mmHg	15.5±5.8	19.4±4.2
Change in PAD (6-month-baseline), mmHg	−3.3±4.3 (P<0.001)	−4.9±4.9 (P=0.002)
6-month Delta HMPI, n (%) [95% CI]	19/29 (65.5) [95% CI 45.7–82.1; P=0.14]	12/15 (80.0)[95% CI 51.9–95.7; P=0.035]

CI, confidence interval; HMPI, hemodynamic management performance index; PAD, pulmonary artery diastolic pressure.
Negative change indicates pulmonary artery diastolic pressure reduction (improved hemodynamic congestion). P-values from paired t-tests within each group.



AC, anticoagulation; ARNi, angiotensin receptor–neprilysin inhibitor; BID, twice daily; BMP, basic metabolic panel; DAPT, dual antiplatelet therapy; GDMT, guideline-directed medical therapy; GFR, glomerular filtration rate; MAG, magnesium; MRA, mineralocorticoid receptor antagonist; MWF, Monday/Wednesday/Friday; PAD, pulmonary artery diastolic pressure; RHC, right heart catheterization; SAPT, single antiplatelet therapy; SGLT2i, sodium-glucose cotransporter-2 inhibitor; Ω, pharmacology trigger criterion.

Figure 1. SMART-HF structured pulmonary artery diastolic pressure–guided workflow. Algorithm diagram illustrating target setting at implant, reassessment during the early post-implant period (Days 5-7), and threshold-based pharmacologic triggers for three pulmonary artery diastolic pressure deviation categories relative to the patient-specific target: ≥5 mmHg below target (de-escalation), 3-5 mmHg above target (moderate escalation), and ≥6 mmHg above target (aggressive escalation). Safety rails include maximum daily loop diuretic doses, mandatory basic metabolic panel and magnesium monitoring, and clinic follow-up triggers.

Baseline characteristics

Baseline characteristics are shown in Table 1.

Primary hemodynamic outcome

In the paired 6-month cohort (n=29), mean baseline PAD was 18.8±6.8 mmHg and mean 6-month PAD was 15.5±5.8 mmHg, corresponding to a mean change of −3.3±4.3 mmHg (95% CI −4.9 to −1.7; P<0.001) (Table 2).

Secondary hemodynamic outcomes

In the paired 90-day cohort (n=36), mean pulmonary artery diastolic pressure decreased from 18.3±7.0 to 16.1±6.3 mmHg (mean change −2.1±3.3 mmHg; 95% CI −3.2 to −1.0; P<0.001). The 90-Day Target HMPI was met in 26/36 (72.2%) [95% CI 54.8–85.8; P=0.011], and a pressure reduction >2 mmHg was observed in 15/36 (41.7%) [95% CI 25.5–59.2; P=0.41]. In the paired 6-month cohort (n=29), the 6-Month Delta HMPI was met in 19/29 (65.5%) [95% CI 45.7-82.1; P=0.14].

Exploratory subgroup analysis: baseline pulmonary artery diastolic pressure >20 mmHg

Nineteen of 37 patients (51.4%) had baseline pulmonary artery diastolic pressure >20 mmHg and comprised the exploratory subgroup. All 19 had paired 90-day windows; 15 had paired 6-month windows. At 90 days (n=19), mean pulmonary artery diastolic pressure decreased from 23.6±3.5 to 20.7±4.2 mmHg (mean change −2.9±3.6 mmHg; 95% CI −4.7 to −1.2; P=0.002). The 90-Day Target HMPI was met in 9/19 (47.4%) [95% CI 24.4-71.1], and a pressure reduction >2 mmHg was observed in 9/19 (47.4%) [95% CI 24.4-71.1]; these proportions were coincidentally identical but did not represent entirely the same patients. At 6 months (n=15), mean pulmonary artery diastolic pressure decreased from 24.3±3.7 to 19.4±4.2 mmHg (mean change −4.9±4.9 mmHg; 95% CI −7.6 to −2.2; P=0.002). The 6-Month Delta HMPI was met in 12/15 (80.0%) [95% CI 51.9-95.7; P=0.035].

Discussion

In this community program, SMART-HF – a structured, algorithm-driven workflow for remote pulmonary artery pressure management – produced statistically significant reductions in ambulatory pulmonary artery diastolic pressure at both 90 days and 6 months. Unlike *ad hoc* approaches in which clinician responses to hemodynamic data vary by provider, day, and institution, SMART-HF standardizes the entire chain of action: target setting, review cadence, threshold-based pharmacologic triggers, and safety monitoring. The result is a safe, reproducible system that can be taught, audited, and scaled – addressing the well-documented implementation gap between the availability of remote hemodynamic data and its translation into consistent, evidence-aligned therapy.[5]

Initial description of the operational framework underpinning SMART-HF was in a 2025 publication. For the first time, a comprehensive unambiguous approach to remote pulmonary artery pressure management had been provided. Every step of the process was detailed, starting with target pressure selection at

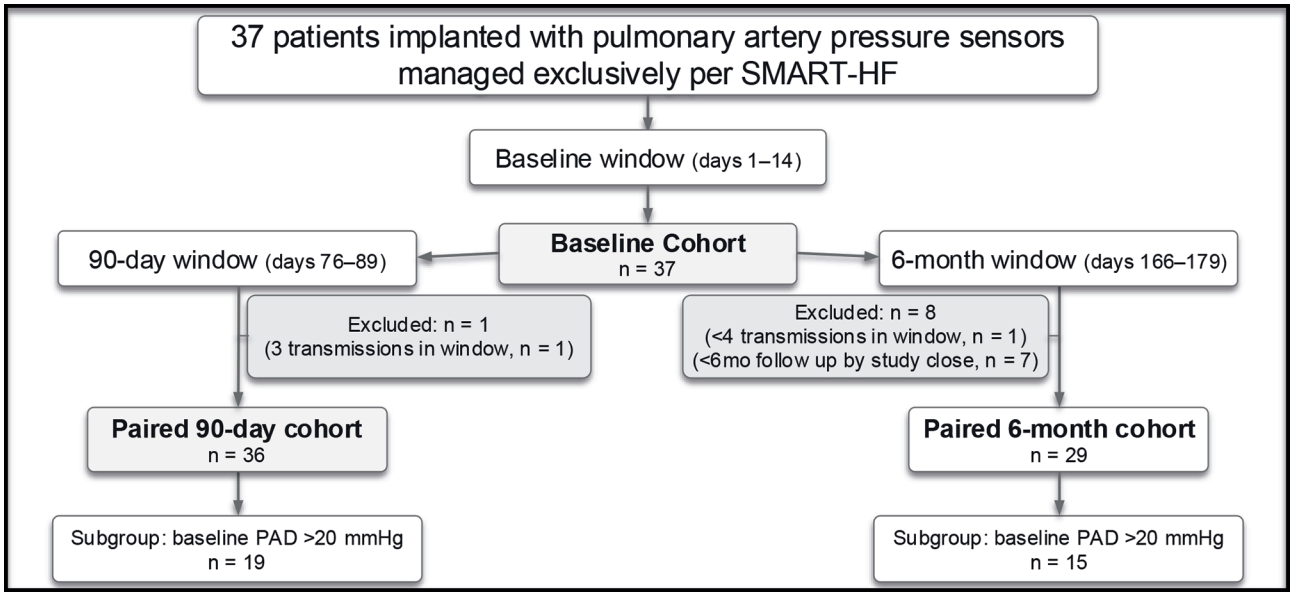


Figure 2. Flow diagram of patient inclusion and analytic cohorts. The 90-day and 6-month windows were assessed independently; patients whose 6-month follow-up window had not been reached by the data lock (1 April 2026) contributed only to the 90-day analysis. Subgroup analyses applied the same paired-window inclusion rule, requiring ≥4 pressure transmissions within each follow-up window.

implantation. The algorithm compelled aggressive early post-implant pressure reduction, further mandating early and frequent reassessment of goal pressures to a target of no more than 15 mmHg. Explicit guidance continued all the way down to patient-specific individualization of hemodynamic triggers and diuretic strategies, all while maintaining safety through prescribed guardrails.[5] The present analysis extends this foundational work by demonstrating that SMART-HF achieves hemodynamic performance benchmarks that have subsequently been independently linked to mortality reduction.[3,4]

Prior publications shared, in the broadest of terms, management guidance for clinicians to reduce filling pressures using diuretics and vasodilators without specifying how, when, or by how much.[6,8] The distinction is beyond semantic – SMART-HF is a detailed field guide for the effective utilization of precision device-guided heart failure therapy, designed for ready adoption – without the need for additional resources – by virtually any cardiology program already offering remote pulmonary artery pressure monitoring.

Two independent large-scale analyses have now established that ambulatory pulmonary artery diastolic pressure trajectories predict all-cause mortality.[3,4] A pooled analysis of pivotal trial cohorts demonstrated that a pressure reduction >2 mmHg from baseline to 6 months conferred a 14.7% decrease in 2-year mortality, whereas an increase >2 mmHg predicted a 26.7% increase.[3] Separately, a national Medicare cohort analysis showed that among patients with elevated baseline pressures (>20 mmHg), only 24% transitioned to acceptable levels (≤20 mmHg) within 90 days under prevailing management; yet those who did experienced significantly improved survival (hazard ratio [HR] 0.72; 95% CI 0.64–0.81; P<0.001).[4] These findings define the hemodynamic management performance indices (HMPI) against which any structured workflow must now be measured. Measured against these benchmarks, SMART-HF achieved materially superior hemodynamic performance (Figure 3). The 90-Day Target HMPI was met in 26/36 (72.2%) of the overall cohort and in 9/19 (47.4%) of patients with baseline pressures >20 mmHg – approximately double the 24% transition rate observed in the Medicare cohort under *ad hoc* management.[4] The 6-Month Delta HMPI was met in 19/29 (65.5%) overall and 12/15

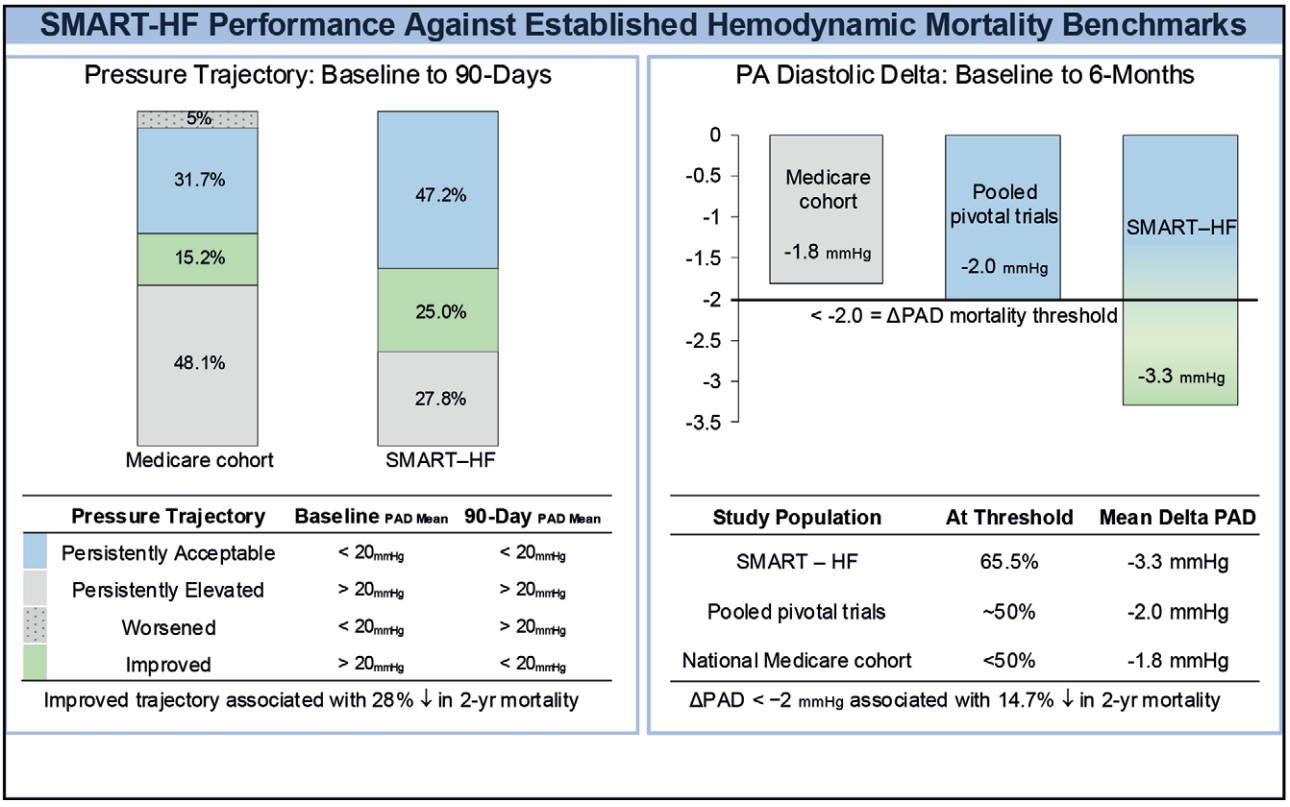


Figure 3. Mortality impact of structured *versus ad hoc* management in hemodynamically monitored heart failure. A) Definitions of pulmonary artery diastolic pressure trajectory categories based on baseline and 90-day status relative to the 20mmHg threshold. B) Distribution of trajectory categories at 90-day follow-up under SMART-HF compared with a national Medicare cohort managed under *ad hoc* protocols; SMART-HF achieved an improved-to-acceptable transition rate approximately double that of the Medicare cohort (47.4% *vs* 24%). C) Mean change in pulmonary artery diastolic pressure at 6 months: SMART-HF (−3.3 mmHg) *versus* Medicare cohort (−1.8 mmHg) *versus* pooled pivotal trial populations (−2.0 mmHg), with the mortality threshold (ΔPAD ≤−2 mmHg) indicated. PAD, pulmonary artery diastolic pressure; HMPI, hemodynamic management performance index.
Data sources: Zile *et al.*, Zalawadiya *et al.*, Atzenhoefer *et al.*[3-5] Analysis time points differ between sources (90 days *vs* 6 months); SMART-HF analyses performed at both time points.

(80.0%) of the elevated-baseline subgroup (P=0.035), with a mean 6-month pressure reduction of −3.3 mmHg across the cohort − nearly double the −1.8 mmHg observed in the Medicare cohort and exceeding the −2.0 mmHg reported in pooled pivotal trial populations.[3,4] Extrapolation of these mortality associations to the present cohort suggests that SMART-HF would confer mortality benefit to a substantially larger proportion of patients than has been achieved under any previously described management approach. The magnitude of pulmonary artery diastolic pressure reduction was greater at 6 months than at 90 days and was greatest among patients with baseline pressures >20 mmHg, suggesting that our structured approach drives continued hemodynamic improvement beyond the early post-implant period. Notably, this cohort − characterized by advanced age and multiple comorbid conditions − represents the patient population frequently cited as frail and difficult to manage with a hemodynamic-guided treatment strategy through remote pulmonary artery pressure monitoring.

Loop diuretics are essential for relief of congestive symptoms but, unlike disease-modifying therapies, have no demonstrated effect on long-term survival.[9] Successive randomized and observational analyses − including a contemporary head-to-head comparison of torsemide and furosemide − have not identified a survival signal attributable to the choice or intensity of loop diuretic therapy.[10-12] A common feature of these studies was that their clinical endpoints relied on late manifestations of hemodynamic congestion − physical examination findings and, in many cases, subjective patient-reported symptoms. It is therefore unsurprising that the agents and doses evaluated were administered without any means of verifying, in real time, whether meaningful hemodynamic impact had been achieved at all. Those investigators conducted the best science possible within the constraints of their epoch; we are now afforded precision methodologies that would have been unfathomable not long ago. In the same spirit of scientific inquiry, the conclusions of that era warrant reconsideration with our tools of greater precision. Indeed, recent landmark trials of hemodynamic-guided management have shown reduced heart-failure hospitalizations, with secondary analyses attributing this benefit to targeted diuretic titration and vasodilator therapy rather than to escalation of disease-modifying guideline-directed medical therapy.[6-8]

Taken together, these observations reframe the diuretic–mortality question. Prior studies asked whether a specific loop diuretic improves survival and found no signal − but none had the ability to measure whether the diuretic actually achieved its hemodynamic objective. SMART-HF demonstrates that when diuretic therapy is titrated with precision against a continuously measured hemodynamic target, pulmonary artery pressures can be reduced reliably and sustainably. The mortality data now available indicate that these pressure reductions − regardless of the specific pharmacologic agent used to achieve them − are independently associated with improved survival.[3,4] The critical variable may not be which diuretic is prescribed, but whether it is dosed and adjusted with sufficient precision to achieve and maintain hemodynamic targets that matter.

Safety

SMART-HF was not associated with any serious adverse event, hospitalization, or complication directly attributable to the workflow. Across the observation period, 15 of 37 patients required additional management for a treatment-related side effect − 7 for electrolyte derangements and 8 for changes in renal function − all of which resolved with standard measures.

Electrolyte derangements

Mild hypokalemia, hyperkalemia, and hypomagnesaemia were encountered. Hyperkalemia related to spironolactone initiation occurred in two patients and resolved on discontinuation. The remaining five occurrences of hypokalemia and hypomagnesaemia were attributed to diuretic-induced renal losses: one patient with loop-diuretic hypokalemia had spironolactone added for correction; two patients had concurrent hypokalemia and hypomagnesaemia attributed to metolazone; and two had isolated hypokalemia or hypomagnesaemia unrelated to metolazone. Oral potassium and magnesium supplementation was sufficient in all cases.

Renal function

Labile renal indices meeting kidney disease: Improving Global Outcomes (KDIGO) criteria for acute kidney injury (AKI) were observed in eight patients, all during active decongestion and all with pulmonary artery diastolic pressures >22 mmHg at the time of creatinine elevation. In five of these eight patients, the underlying etiology was occult paroxysmal atrial fibrillation (Section 4.2). The remaining three patients were referred to nephrology: two had diabetic chronic kidney disease in whom continued reduction of pulmonary artery diastolic pressures did not result in further elevation of serum creatinine, and one had acute tubular necrosis from hypotension attributed to combination guideline-directed medical therapy, with serum creatinine returning to baseline after discontinuation of the sodium-glucose cotransporter 2 (SGLT2) inhibitor and dose reduction of sacubitril-valsartan.

Pressure waveforms reveal pathology beyond congestion

Beyond quantifying congestion, transmitted pulmonary artery pressure waveforms isolated arrhythmia as a significant driver of pressure elevation, heart failure symptoms, and a barrier to successful decongestion which the conventional bedside assessment of fluid-status would not detect. Among the eight patients who developed acute kidney injury during active decongestion, five were found to have previously undiagnosed paroxysmal atrial fibrillation. Labile PA pressures associated with changes in heart rate signaled closer examination of their transmitted pressure waveforms which were morphologically abnormal. Subsequent ambulatory electrocardiographic monitoring confirmed the paroxysmal atrial

fibrillation, for which oral antiarrhythmic therapy was pursued with or without external direct current cardioversion. Restoration and maintenance of sinus rhythm preceded precipitous falls in pulmonary artery diastolic pressures alongside stabilization of renal function without additional diuretic escalation. This observation is clinically meaningful in a cohort where 24 of 37 patients (64.9%) carried the comorbid diagnosis of atrial fibrillation or flutter. In heart failure, occult atrial arrhythmia – clinically silent yet hemodynamically active – is common and consequential; in these patients it was the principal barrier to lowering PAD towards thresholds associated with improved 2-year survival. Beyond the numerical pressure value, waveform morphology holds diagnostic value in the appropriate clinical context. Pattern isolation and algorithmic thresholds could serve to screen for and identify patients with likely occult atrial arrhythmia, prompting additional evaluation. Prompt structured review of transmitted waveforms with timely additional testing and/or specialist referral could reduce the risk of downstream cardioembolic complication and guard against renal injury. Although exploratory and based on small numbers, this finding illustrates a benefit of hemodynamic monitoring that extends beyond the pressure-lowering outcomes captured by our primary analysis and warrants additional study.

Limitations

This single-center study is modest in size (n=37). Incomplete paired follow-up was present for one patient at 90 days and eight patients at 6 months, though the reasons for each are known. The single patient missing from the 90-day comparison did not meet the 4 or more-measurement transmission requirement during the 14-day follow-up window per previously published criteria.[3] The eight patients without 6-month paired data had been implanted fewer than six months before data extraction. Although this missingness is non-informative, it has the potential to introduce bias. No contemporaneous control group was studied in parallel; this is only marginally problematic, as prior robust large-scale trials have rigorously assessed hemodynamic heart failure management *versus* usual care in a prospective randomized fashion, with consistent results. Nevertheless, our results should be interpreted for feasibility and safety rather than causal proof of superiority of SMART-HF over other management approaches.

Conclusions

SMART-HF, a structured pulmonary artery diastolic pressure–guided workflow first described in detail in 2025,[5] produced statistically significant pressure reductions and exceeded independently validated hemodynamic management performance indices at both 90 days and 6 months in a community heart failure cohort characterized by advanced age and multimorbidity. Performance against these mortality-linked benchmarks was materially superior to that observed in both a national Medicare cohort and pooled pivotal trial populations under *ad hoc* management.[3,4] These findings support the adoption of structured, algorithmic hemodynamic management as the operational standard for remote pulmonary artery pressure monitoring programs. Adequately powered comparative trials are warranted to confirm whether the hemodynamic advantages demonstrated here translate into the survival benefit that current mortality data would predict.

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Author contributions

Marc Atzenhoefer: conceptualization, methodology, investigation, supervision, writing original draft, writing-review and editing. Tamar Atzenhoefer: methodology, formal analysis, writing-review and editing. Hussain Boxwala: software, statistical and formal analysis, visualization, writing-review and editing. Brooke Nelson: investigation, resources, writing-review and editing. Michelle Staudacher: investigation, data curation, writing-review and editing. Fahad Iqbal: investigation, writing-review and editing.

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Ethics approval

The study was reviewed by the local institutional review board, which waived informed consent for this retrospective analysis conducted in accordance with the Declaration of Helsinki.

Conflict of interest

Marc Atzenhoefer is a consultant and speaker for Abbott Cardiovascular as a regional Key Opinion Leader for the CardioMEMS HF System. Brooke Nelson is employed by Abbott Cardiovascular, Heart Failure Division, as a clinical support representative. Tamar Atzenhoefer, Hussain Boxwala, Michelle Staudacher, and Fahad Iqbal report no conflicts of interest.

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