Scientific Research Publishing

World Journal of Cardiovascular Diseases, 2026, 16(3), 177-181
https://www.scirp.org/journal/wjcd
ISSN Online: 2164-5337
ISSN Print: 2164-5329

Rethinking Fluid Management in Congestive Heart Failure: Cardiovascular-Renal Regulation, Fluid Management and Diuretics

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How to cite this paper: Wang, D.Y. (2026) Rethinking Fluid Management in Congestive Heart Failure: Cardiovascular-Renal Regulation, Fluid Management and Diuretics. *World Journal of Cardiovascular Diseases*, **16**, 177-181.
https://doi.org/10.4236/wjcd.2026.163018

Received: January 21, 2026
Accepted: March 9, 2026
Published: March 12, 2026

Abstract

Despite substantial advances in guideline-directed medical therapy, recurrent hospitalization remains a persistent challenge in the management of congestive heart failure (CHF). Fluid overload is commonly treated as the primary driver of decompensation, with chronic diuretic therapy serving as the cornerstone of outpatient management. However, many patients admitted with CHF exacerbation are already receiving escalating doses of diuretics, raising important questions regarding the underlying mechanisms of recurrent decompensation. This perspective proposes a systems-based, hypothesis-generating framework that reframes congestion as a downstream manifestation of impaired cardiovascular-renal regulation rather than a primary disorder of fluid excess. We examine how rigid, protocol-driven approaches to diuretic use may disrupt physiological equilibrium in susceptible patients and contribute to instability, neurohormonal overactivation, and readmission. We further discuss practice gaps in outpatient fluid management, including inconsistent application of fluid restriction and limited attention to individual tolerance. Finally, we outline the rationale for an individualized, patient-centered approach to fluid management that complements existing guideline-based therapies and warrants prospective investigation.

Keywords

Cardiovascular-Renal System, Fluid Management, Diuretics

1. Introduction

Congestive heart failure remains a leading cause of hospitalization and readmission worldwide, imposing substantial clinical and economic burdens [1]-[3]. Alt-

hough modern guideline-directed medical therapy (GDMT) has improved survival and long-term outcomes, rates of recurrent decompensation remain high [4] [5]. A striking paradox in contemporary practice is that the majority of patients admitted for CHF exacerbation are already receiving chronic diuretic therapy, often at increasing doses, yet continue to experience congestion and clinical instability [6]-[9].

Traditionally, CHF decompensation has been conceptualized as a problem of fluid overload, with diuretics employed to restore euvolemia. While this approach is effective for symptomatic relief in acute settings, it may oversimplify the underlying pathophysiology in the outpatient context. Increasing evidence and clinical observation suggest that congestion frequently reflects dysfunction of the cardiovascular-renal regulatory system rather than simply absolute excess of total body fluid, with fluid accumulation representing a downstream manifestation of this dysfunction.

This perspective argues that chronic, rigid approaches to fluid management—particularly routine daily diuretic use combined with poorly individualized fluid intake—may destabilize cardiovascular-renal homeostasis in selected patients. We propose that a more nuanced, patient-specific strategy is needed, one that recognizes the dynamic nature of fluid balance, inter-individual variability in tolerance, and the central role of cardiovascular-renal regulation in maintaining equilibrium of the fluid metabolism.

2. Cardiovascular-Renal Regulation and Fluid Homeostasis

Fluid balance in humans is governed by a tightly coupled cardiovascular-renal system designed to maintain adequate intravascular volume, tissue perfusion, and blood pressure across a wide range of daily conditions. This system continuously adjusts renal perfusion, sodium handling, and urine output in response to fluctuations in intake, activity, and hemodynamic demand. Importantly, each individual operates within a unique physiologic "operating range" that reflects cardiac reserve, renal function, neurohormonal tone, age, and comorbid conditions in this individual.

In CHF, this operating range becomes narrowed. Structural or functional cardiac abnormalities, renal impairment, and heightened neurohormonal activation reduce tolerance to both volume expansion and depletion. Under these conditions, small perturbations in intake or output may result in disproportionate clinical consequences.

Diuretics, while effective at promoting natriuresis and relieving congestion, act as fixed external perturbations within this adaptive system. Unlike endogenous regulatory mechanisms, their effects are not dynamically adjusted to moment-to-moment physiologic needs. In certain patients, chronic diuretic exposure may therefore predispose to intravascular volume depletion, reduced renal perfusion, and compensatory neurohormonal activation, especially when fluid intake decreases or fluid loss increases, such as with diarrhea. Paradoxically, this state may

impair effective fluid excretion and promote fluid redistribution into the interstitial space, manifesting clinically as pulmonary congestion and peripheral edema. This response may amplify the clinical picture of fluid overload, often promoting escalation of diuretic therapy, which may further perturb cardiovascular-renal dysfunction and contribute to progressive congestion and hospitalization.

3. Practice Gaps in Outpatient Fluid Management

Current outpatient CHF management frequently emphasizes pharmacologic therapy while devoting comparatively less attention to individualized fluid intake. In real-world practice, fluid restriction is inconsistently applied, variably emphasized by clinicians, and often poorly adhered to by patients [10]. In some settings, liberal fluid intake is implicitly permitted or even encouraged, with the assumption that excess volume can be managed through diuretic escalation.

This reactive paradigm places increasing reliance on diuretics to correct physiologic imbalance after it has already occurred. However, excessive or poorly matched diuretic use may further narrow the cardiovascular-renal operating range, amplify neurohormonal activation, and limit tolerance to everyday fluctuations in intake. The result may be a self-perpetuating cycle of diuretic escalation, renal dysfunction, and recurrent congestion.

Importantly, tolerance to both fluid intake and diuretic exposure varies widely among patients. Uniform recommendations for stricter fluid restriction or routine daily diuretic use may therefore stabilize some individuals while destabilizing others. These observations highlight a critical gap between guideline-level recommendations and the individualized application required in everyday clinical care.

4. The Case for Individualized, Patient-Centered Fluid Management

We propose that outpatient fluid management in CHF should be reframed as an individualized, patient-centered process rather than a fixed protocol. Such an approach begins with recognition that fluid balance is dynamic and that each patient has a unique physiologic tolerance determined by cardiovascular-renal function, lifestyle, and comorbidities.

Individualized fluid management involves defining a flexible intake range tailored to the patient's tolerance rather than imposing rigid universal limits. Diuretics are reserved for periods when physiologic capacity is exceeded, rather than employed routinely as a default. Clinical assessment integrates daily weight trends, symptoms, functional capacity, and renal parameters to guide adjustment over time.

This strategy does not negate the importance of GDMT or the role of diuretics in acute decompensation. Rather, it seeks to align outpatient management more closely with underlying physiology, minimizing unnecessary perturbation of cardiovascular-renal regulation while preserving the ability to intervene when needed.

5. Implications and Future Directions

The framework outlined here is intended to be hypothesis-generating rather than prescriptive, although some clinical experience and prior literature provide contextual support for the framework [11]. It raises the possibility that recurrent CHF decompensation in a subset of patients may be driven, at least in part, by maladaptive fluid management strategies rather than inexorable disease progression alone.

Prospective studies are needed to evaluate whether individualized fluid restriction combined with selective, as-needed diuretic use can reduce instability and readmission while maintaining symptom control and adherence to GDMT. Such investigations may also clarify which patient phenotypes are most likely to benefit from this approach [12] [13].

By shifting focus from rigid protocols to physiologic regulation and patient-specific tolerance, individualized fluid management offers a potential avenue to improve outcomes in a population that continues to experience high morbidity despite therapeutic advances, highlighting the importance of individualized clinical judgment alongside evidence-based therapy.

6. Conclusion

Recurrent decompensation in congestive heart failure remains a major unmet challenge. Viewing congestion as a manifestation of impaired cardiovascular-renal regulation rather than simple fluid excess may help explain why many patients deteriorate despite chronic diuretic therapy. An individualized, patient-centered approach to fluid management that respects physiologic variability and minimizes unnecessary perturbation represents a rational, testable strategy that warrants further investigation.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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