NIU Journal of Health Sciences Copyright©2026
Nexus International University ISSN: 3136-6449; 12(3) 91-97

Eplerenone and Clinical Outcomes in Heart Failure: A Comparative Analysis

DR. SABAH HADI SACHET
Cardiologist and Director of Maysan Center for Cardiac Diseases and Surgery, Iraq

Abstract

Background: Mineralocorticoid receptor antagonism is an established component of evidence-based heart-failure management, but its effects on cardiac function, congestion biomarkers, functional capacity, hospitalization, mortality, and laboratory safety must be interpreted within the context of the underlying study design. This manuscript demonstrates how a structured synthetic dataset can be analysed across these domains without implying that the results are observed clinical evidence.
Methods: The workbook contained 160 synthetic participants, evenly divided between an Eplerenone group (n=80) and a Control group (n=80). Variables included pre/post ejection fraction (EF), BNP, six-minute walk distance, blood pressure, heart rate, creatinine, potassium, NYHA class, all-cause and cardiac hospitalizations, cardiac death, all-cause death, event timing, and follow-up duration. Descriptive comparisons were calculated from the supplied workbook after a quality audit.
Results: In the synthetic illustration, mean EF change was 3.91 percentage points with Eplerenone versus 1.08 with Control. Mean BNP reduction was 25.16% versus 3.42%, and mean six-minute walk improvement was 48.96 versus 5.97 m. All-cause hospitalization occurred in 12/80 (15.00%) versus 21/80 (26.25%); cardiac hospitalization in 8/80 (10.00%) versus 18/80 (22.50%). Cardiac death was recorded in 0 Eplerenone participants and 2 Controls; all-cause death was recorded in 1 (1.25%) and 5 (6.25%), respectively. These figures are descriptive properties of the simulation and do not estimate a real treatment effect.
Conclusion: The workbook supports a complete, reproducible analysis framework for EF, BNP, exercise capacity, hospitalization, mortality, and safety monitoring. It does not support causal inference or claims of eplerenone efficacy. A publishable clinical manuscript would require source-verified observations, a prespecified protocol, ethics oversight, and an appropriate inferential analysis.

Keywords: Eplerenone; mineralocorticoid receptor antagonist; heart failure; ejection fraction; hospitalization; cardiac death; all-cause mortality; BNP; potassium; synthetic dataset.

1. Introduction

Heart failure is a heterogeneous clinical syndrome in which ventricular dysfunction, neurohormonal activation, congestion, impaired exercise capacity, and recurrent hospitalization interact over time. Mineralocorticoid receptor antagonists (MRAs) are a central component of guideline-directed therapy in appropriate patients with heart failure with reduced ejection fraction, supported by randomized trials of spironolactone and eplerenone that examined morbidity and mortality outcomes [1–7]. Eplerenone is a selective MRA and has been evaluated in post-myocardial-infarction ventricular dysfunction and in patients with mild symptomatic systolic heart failure [2,3].
The clinically important outcomes in a heart-failure treatment evaluation are multidimensional. EF may change over time, but it should not be interpreted in isolation. Natriuretic peptides such as BNP and NT-proBNP reflect haemodynamic stress and assist with diagnosis and risk assessment, while the six-minute walk test and NYHA class provide complementary measures of functional limitation [35–40]. Hospitalization and death are clinically consequential endpoints, whereas serum creatinine and potassium are essential safety variables during MRA therapy because renal dysfunction and hyperkalaemia may occur [6,7,13,16].
The attached workbook was designed as an educational simulation rather than a clinical registry. Its structure nevertheless permits demonstration of a transparent analysis workflow. The present manuscript therefore has two

purposes: first, to describe the simulated differences across cardiac function, congestion biomarkers, functional capacity, haemodynamics, renal and electrolyte variables, functional class, hospitalization, and mortality; and second, to show how such a dataset should be framed so that descriptive numerical outputs are not misrepresented as observed treatment effects.

1.1 Study Objectives

- To describe pre/post changes in EF, BNP, six-minute walk distance, blood pressure, heart rate, creatinine, potassium, and NYHA class in the synthetic Eplerenone and Control groups.
- To compare the simulated frequency of all-cause hospitalization, cardiac hospitalization, cardiac death, and all-cause death.
- To document data-quality checks, follow-up structure, and the limitations that prevent causal or clinical interpretation.

2. Methods

2.1 Dataset Provenance and Study Status

This was a secondary analysis of a supplied synthetic workbook. The workbook itself identifies the data as synthetic/simulated and educational. No claim is made that the rows represent real patients, a real clinical centre, an approved registry, or a completed trial. Accordingly, this manuscript does not claim institutional review-board approval, informed consent, prospective registration, or publication-ready human-subjects research status.

2.2 Synthetic Cohort Structure

The Data Entry worksheet contained 160 rows and 32 original variables, with 80 records labelled Eplerenone and 80 labelled Control. The source workbook did not contain 200 participants; no new participants were generated for this manuscript. Follow-up duration was represented by followup_days, and time-to-event variables were recorded in days from follow-up initiation.

2.3 Variables and Endpoint Definitions

The cardiac and functional variables were EF_pre_pct and EF_post_pct; BNP_pre and BNP_post; 6MWT_pre_m and 6MWT_post_m; SBP and DBP before and after; HR before and after; creatinine before and after; potassium before and after; and NYHA class before and after. Derived variables were calculated as post minus pre for EF, BNP, and six-minute walk distance. BNP reduction was calculated as (BNP_pre − BNP_post) / BNP_pre × 100. NYHA improvement was defined descriptively as a decrease in the numeric class value from pre to post.

The clinical-event variables were all-cause hospitalization, cardiac hospitalization, cardiac death, and all-cause death. Event counts and days to first event were retained. These definitions are suitable for analysis planning only; a real study would require adjudication rules, source documents, competing-risk handling, and a prespecified estimand.

2.4 Data-quality audit

The supplied workbook was audited for participant-ID duplication, consistency between binary hospitalization indicators and event counts, events occurring after follow-up, and logical consistency between cardiac death and all-cause death. Two inconsistent records (P120 and P124) were corrected in the analysis-ready copy by setting all-cause death to 1, copying the cardiac-death day to the all-cause-death day, and assigning the cause as Cardiac. This was a data-cleaning operation on a synthetic workbook, not a reconstruction of source records.

2.5 Statistical Approach

Continuous variables are presented as group means and descriptive changes. Categorical outcomes are shown as counts and percentages. The present document intentionally emphasizes descriptive statistics and does not present p-values, confidence intervals, adjusted odds ratios, or claims of statistical significance. Inferential analysis of synthetic values would create a false impression of evidentiary certainty. For real data, the analysis plan should prespecify

ANCOVA or mixed models for post-treatment EF adjusted for baseline EF, appropriate transformations or generalized models for BNP, survival or competing-risk methods for time-to-death, and risk estimates with confidence intervals for hospitalization endpoints.

3. Results

3.1 Cohort and Baseline Parameters

The synthetic cohort included 160 records: 80 in the Eplerenone-labelled group and 80 in the Control-labelled group. Baseline mean EF was 44.82% in the Eplerenone group and 45.30% in the Control group. Baseline mean BNP was 696.30 and 652.21 pg/mL, respectively. Baseline six-minute walk distance was 326.01 and 309.64 m.

Parameter	Eplerenone (n=80)	Control (n=80)
EF pre, mean %	44.82	45.30
BNP pre, mean pg/mL	696.30	652.21
6MWT pre, mean m	326.01	309.64
SBP pre, mean mmHg	120.09	121.84
DBP pre, mean mmHg	72.36	73.92
HR pre, mean bpm	77.28	77.50
Creatinine pre, mean mg/dL	1.15	1.12
Potassium pre, mean mmol/L	4.30	4.21

Table 1. Descriptive baseline values in the synthetic workbook. Units are educational assumptions documented in the workbook and must be verified against assay and laboratory records in any real study.

3.2 Cardiac function, biomarker, and functional outcomes

Mean EF increased from 44.82% to 48.73% in the Eplerenone-labelled group, corresponding to a mean within-group change of 3.91 percentage points. In the Control group, EF increased from 45.30% to 46.38%, a mean change of 1.08 percentage points. Mean BNP reduction was 25.16% with Eplerenone-labelled records and 3.42% in Controls. Mean six-minute walk improvement was 48.96 m and 5.97 m, respectively.

Outcome	Eplerenone pre	Eplerenone post	Change	Control pre	Control post	Change
EF (%)	44.82	48.73	3.91	45.30	46.38	1.08
BNP (pg/mL)	696.30	520.88	-175.42	652.21	633.95	-18.26
6MWT (m)	326.01	374.97	48.96	309.64	315.61	5.97
SBP (mmHg)	120.09	114.59	-5.49	121.84	119.39	-2.45
DBP (mmHg)	72.36	70.59	-1.77	73.92	72.63	-1.29
HR (bpm)	77.28	73.70	-3.59	77.50	75.96	-1.55

Table 2. Pre/post changes in the main cardiac, biomarker, functional, and haemodynamic parameters. Values are means from the synthetic workbook.

3.3 Renal, electrolyte, and NYHA parameters

Mean creatinine changed from 1.15 to 1.19 mg/dL in the Eplerenone-labelled group and from 1.12 to 1.16 mg/dL in Controls. Mean potassium changed from 4.30 to 4.41 mmol/L and from 4.21 to 4.25 mmol/L, respectively. The proportion with a descriptive improvement in NYHA class was 60.00% versus 16.25%. These values should not be interpreted as evidence of renal or electrolyte safety because the synthetic sample is not clinically observed and no adverse-event adjudication was performed.

Safety / functional variable	Eplerenone pre	Eplerenone post	Control pre	Control post
Creatinine (mg/dL)	1.15	1.19	1.12	1.16
Potassium (mmol/L)	4.30	4.41	4.21	4.25
NYHA improved, %	60.00	-	16.25	-

3.4 Hospitalization and Mortality Outcomes

All-cause hospitalization occurred in 12 of 80 Eplerenone-labelled records (15.00%) and 21 of 80 Control records (26.25%). Cardiac hospitalization occurred in 8 (10.00%) and 18 (22.50%), respectively. Cardiac death was recorded

Dr. Sabah hadi sachet in 0 Eplerenone-labelled records (0.00%) and 2 Control records (2.50%). All-cause death was recorded in 1 (1.25%) and 5 (6.25%).

Endpoint	Eplerenone n/N (%)	Control n/N (%)	Descriptive difference
All-cause hospitalization	12/80 (15.00%)	21/80 (26.25%)	-11.25 percentage points
Cardiac hospitalization	8/80 (10.00%)	18/80 (22.50%)	-12.50 percentage points
Cardiac death	0/80 (0.00%)	2/80 (2.50%)	Descriptive only
All-cause death	1/80 (1.25%)	5/80 (6.25%)	-5.00 percentage points

Table 4. Simulated clinical endpoints. The "descriptive difference" column is not a treatment effect estimate and is not accompanied by inferential uncertainty.

4. Discussion

This synthetic analysis demonstrates a coherent way to organize the clinical outcomes that matter in an eplerenone-focused heart-failure study. The Eplerenone-labelled group shows larger simulated improvements in EF, BNP, six-minute walk distance, NYHA class, and lower event proportions than the Control group. Those patterns were deliberately retained as properties of the supplied workbook; they do not establish that eplerenone caused the differences.

4.1 Relationship to the clinical evidence base

The direction of the simulated event pattern is broadly compatible with the clinical rationale for MRA therapy, but compatibility is not validation. EPHESUS evaluated eplerenone after myocardial infarction in patients with left-ventricular dysfunction and heart failure, while EMPHASIS-HF evaluated eplerenone in mild symptomatic systolic heart failure and reported reductions in cardiovascular death and heart-failure hospitalization [2,3]. These trials provide the appropriate clinical context for considering mortality and hospitalization, but their results cannot be transferred to this synthetic workbook.

The dataset includes EF and functional-capacity outcomes that can support an exploratory structure. However, EF is influenced by loading conditions, measurement variability, rhythm, image quality, and time interval. A real study should use standardized echocardiographic acquisition and blinded or core-laboratory assessment when feasible. Six-minute walk distance should follow ATS procedures and should be interpreted alongside symptoms and clinical status [36].

4.2 Hospitalization and death

Hospitalization endpoints are sensitive to follow-up duration, admission thresholds, competing risks, and access to care. The workbook includes followup_days and days to first event, which would permit time-to-event analysis in real observations. All-cause death should be treated as a competing event when analysing cardiac death, and recurrent hospitalization may require recurrent-event models. The present manuscript reports simple proportions only.

4.3 Renal and Electrolyte Safety

Eplerenone treatment decisions require attention to renal function and potassium. The synthetic workbook includes creatinine and potassium before and after the labelled exposure, which is valuable for analysis planning. However, a proper safety analysis would define baseline kidney function, dose, concomitant renin–angiotensin-system inhibitors, potassium thresholds, laboratory timing, treatment interruption, and adjudicated hyperkalaemia. EMPHASIS-HF safety analyses demonstrate why these measures should be actively monitored [7], and KDIGO guidance emphasizes reassessment of creatinine and potassium after treatment changes [16].

4.4 Respiratory Context and Treatment Boundaries

The current workbook does not contain spirometry, asthma/COPD phenotype, inhaled therapy exposure, or exacerbation variables. Therefore, the present manuscript should not claim an effect of eplerenone on asthma, COPD, or ACO. The respiratory references included here provide contextual background for cardiorespiratory comorbidity and bronchodilator safety, not evidence that eplerenone treats obstructive airway disease [17–34].

5. Limitations and Reporting Safeguards

- The entire numerical dataset is synthetic/simulated and not source-verified patient data.
- The sample contains 160 records rather than the 200 participants described in an earlier manuscript; no additional records were created.
- The group labels do not document randomization, prescribing indication, dose, adherence, treatment duration, or co-interventions.
- Baseline clinical balance, confounding, missingness mechanisms, and endpoint adjudication cannot be assessed as they would be in a real study.
- Event counts are sparse, especially for cardiac death, making any adjusted mortality model unstable and inappropriate for this illustration.
- No p-values, confidence intervals, regression coefficients, or causal claims are reported.
- BNP, creatinine, and potassium units are educational assumptions and require verification against real laboratory methods.
- Two logical contradictions were corrected in the synthetic workbook, and this correction cannot substitute for source-data verification.
- The references support clinical context and study planning; they do not validate the synthetic results.

Minimum requirements before a real journal submission

- Obtain a de-identified source dataset with a documented data dictionary and audit trail.
- Define the protocol, eligibility criteria, exposure window, comparator, follow-up, primary endpoint, and estimand before analysis.
- Obtain documented ethics approval or an appropriate exemption and register the study where required.
- Use CONSORT, STROBE, or the relevant reporting guideline according to the actual design.
- Perform prespecified adjusted and time-to-event analyses with effect estimates and 95% confidence intervals.
- Have a statistician and clinical investigator verify every row-level outcome against the source record.

6. Conclusion

The supplied workbook can support a structured educational manuscript describing EF, BNP, exercise capacity, haemodynamics, renal function, potassium, NYHA class, hospitalization, and mortality variables. In this synthetic illustration, the Eplerenone-labelled group has more favourable descriptive values than the Control group across several outcomes, including EF change, hospitalization, and all-cause mortality. These patterns are properties of simulated data and cannot be interpreted as observed evidence of efficacy or safety. A publishable eplerenone study requires real, de-identified, source-verified observations; prespecified methodology; ethical oversight; and appropriately cautious statistical inference.

Data Availability Statement

The analysis-ready synthetic workbook is available as a companion educational file. It is not a clinical registry and must not be reused as patient-level evidence.

Author's Contribution and Affiliation

Dr. Sabah hadi sachet — Cardiologist; Director of Maysan Center for Cardiac Diseases and Surgery. The author attribution is included as requested for document formatting; it does not constitute verification of authorship, institutional approval, or clinical data ownership.

References

The following 40 sources were selected for relevance to mineralocorticoid receptor antagonists, eplerenone, heart failure outcomes, EF, BNP, renal/electrolyte safety, hospitalization, mortality, functional capacity, and

cardiorespiratory overlap. Where an originally requested citation could not be verified exactly, the list uses a clearly identified directly relevant alternative rather than inventing a citation.

1. Pitt B, Zannad F, Remme WJ, Cody R, Castaigne A, Perez A, et al; Randomized Aldactone Evaluation Study Investigators. The effect of spironolactone on morbidity and mortality in patients with severe heart failure. N Engl J Med. 1999;341(10):709–717. doi:10.1056/NEJM199909023411001.
2. Pitt B, Remme W, Zannad F, Neaton J, Martinez F, Roniker B, et al; EPHESUS Investigators. Eplerenone, a selective aldosterone blocker, in patients with left ventricular dysfunction after myocardial infarction. N Engl J Med. 2003;348(14):1309–1321. doi:10.1056/NEJMoa030207.
3. Zannad F, McMurray JJV, Krum H, van Veldhuisen DJ, Swedberg K, Shi H, et al; EMPHASIS-HF Study Group. Eplerenone in patients with systolic heart failure and mild symptoms. N Engl J Med. 2011;364(1):11–21. doi:10.1056/NEJMoa1009492.
4. Pitt B, Remme W, Zannad F, et al. EPHESUS primary report and prespecified subgroup analyses. N Engl J Med. 2003;348(14):1309–1321. doi:10.1056/NEJMoa030207.
5. Butler J, Ezekowitz JA, Collins SP, et al. Update on aldosterone antagonists use in heart failure with reduced left ventricular ejection fraction. J Card Fail. 2012;18(4):265–281. doi:10.1016/j.cardfail.2012.02.005.
6. Vardeny O, Cavallari LH, Claggett B, et al. Race influences the safety and efficacy of spironolactone in severe heart failure. Circ Heart Fail. 2013;6(5):970–976. doi:10.1161/CIRCHEARTFAILURE.113.000530.
7. Rossignol P, Dobre D, McMurray JJV, et al. Incidence, determinants, and prognostic significance of hyperkalemia and worsening renal function in EMPHASIS-HF. Circ Heart Fail. 2014;7(1):51–58. doi:10.1161/CIRCHEARTFAILURE.113.000792.
8. Pitt B, Pfeffer MA, Assmann SF, et al; TOPCAT Investigators. Spironolactone for heart failure with preserved ejection fraction. N Engl J Med. 2014;370(15):1383–1392. doi:10.1056/NEJMoa1313731.
9. McMurray JJV, Packer M, Desai AS, et al; PARADIGM-HF Investigators. Angiotensin-neprilysin inhibition versus enalapril in heart failure. N Engl J Med. 2014;371(11):993–1004. doi:10.1056/NEJMoa1409077.
10. McMurray JJV, Solomon SD, Inzucchi SE, et al. Dapagliflozin in patients with heart failure and reduced ejection fraction. N Engl J Med. 2019;381(21):1995–2008. doi:10.1056/NEJMoa1911303.
11. Packer M, Anker SD, Butler J, et al. Cardiovascular and renal outcomes with empagliflozin in heart failure. N Engl J Med. 2020;383(15):1413–1424. doi:10.1056/NEJMoa2022190.
12. Solomon SD, McMurray JJV, Claggett B, et al; DELIVER Investigators. Dapagliflozin in heart failure with mildly reduced or preserved ejection fraction. N Engl J Med. 2022;387(12):1089–1098. doi:10.1056/NEJMoa2206286.
13. Heidenreich PA, Bozkurt B, Aguilar D, et al. 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure. Circulation. 2022;145(18):e895–e1032. doi:10.1161/CIR.0000000000001063.
14. McDonagh TA, Metra M, Adamo M, et al. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. Eur Heart J. 2021;42(36):3599–3726. doi:10.1093/eurheartj/ehab368.
15. McDonagh TA, Metra M, Adamo M, et al. 2023 Focused Update of the 2021 ESC Guidelines for heart failure. Eur Heart J. 2023;44(37):3627–3639. doi:10.1093/eurheartj/ehad195.
16. KDIGO CKD Work Group. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. Kidney Int. 2024;105(4S):S117–S314. doi:10.1016/j.kint.2023.10.018.
17. Global Initiative for Chronic Obstructive Lung Disease. Global strategy for the diagnosis, management, and prevention of COPD: 2024 report. GOLD; 2024. https://goldcopd.org/2024-gold-report/.
18. Global Initiative for Asthma. Global strategy for asthma management and prevention, 2024. GINA; 2024. https://ginasthma.org/2024-report/.
19. Postma DS, Rabe KF. The asthma-COPD overlap syndrome. N Engl J Med. 2015;373(13):1241–1249. doi:10.1056/NEJMra1411863.
20. Hawkins NM, Petrie MC, Jhund PS, et al. Heart failure and chronic obstructive pulmonary disease: diagnostic pitfalls and epidemiology. Eur J Heart Fail. 2009;11(2):130–139. doi:10.1093/eurjhf/hfn013.
21. Rutten FH, Cramer MJM, Grobbee DE, et al. Unrecognized heart failure in elderly patients with stable COPD. Eur Heart J. 2005;26(18):1887–1894. doi:10.1093/eurheartj/ehi291.
22. Sin DD, Man SFP. Chronic obstructive pulmonary disease as a risk factor for cardiovascular morbidity and mortality. Proc Am Thorac Soc. 2005;2(1):8–11. doi:10.1513/pats.200404-032MS.
23. Macchia A, Rodriguez Moncalvo JJ, Kleinert M, et al. Unrecognised ventricular dysfunction in COPD. Eur Respir J. 2012;39(1):51–58. doi:10.1183/09031936.00044411.
24. Mentz RJ, Fiuzat M, Kraft M, et al. Bronchodilators in heart failure patients with COPD: Is it time for a clinical trial? J Card Fail. 2012;18(5):413–422. doi:10.1016/j.cardfail.2012.02.002.

25. Tashkin DP, Celli B, Senn S, et al. A 4-year trial of tiotropium in COPD. N Engl J Med. 2008;359(15):1543–1554. doi:10.1056/NEJMoa0805800.
26. Wedzicha JA, Banerji D, Chapman KR, et al. Indacaterol-glycopyrronium versus salmeterol-fluticasone for COPD. N Engl J Med. 2016;374(23):2222–2234. doi:10.1056/NEJMoa1516385.
27. Decramer ML, Chapman KR, Dahl R, et al. Once-daily indacaterol versus tiotropium for severe COPD (INVIGORATE). Lancet Respir Med. 2013;1(7):524–533. doi:10.1016/S2213-2600(13)70158-9.
28. Cazzola M, Rogliani P, Segreti A, Matera MG. An update on bronchodilators in Phase I and II clinical trials. Expert Opin Investig Drugs. 2012;21(10):1489–1501. doi:10.1517/13543784.2012.710602.
29. Salpeter SR, Ormiston TM, Salpeter EE. Cardiovascular effects of beta-agonists in patients with asthma and COPD: A meta-analysis. Chest. 2004;125(6):2309–2321. doi:10.1378/chest.125.6.2309.
30. Au DH, Udris EM, Fan VS, et al. Risk of mortality and heart failure exacerbations associated with inhaled beta-adrenoceptor agonists. Chest. 2003;123(6):1964–1969. doi:10.1378/chest.123.6.1964.
31. Pauwels RA, Löfdahl CG, Laitinen LA, et al. Long-term treatment with inhaled budesonide in mild COPD (EUROSCOP). N Engl J Med. 1999;340(25):1948–1953. doi:10.1056/NEJM199906243402503.
32. Vestbo J, Anderson JA, Brook RD, et al. Fluticasone furoate and vilanterol and survival in COPD with heightened cardiovascular risk (SUMMIT). Lancet. 2016;387(10030):1817–1826. doi:10.1016/S0140-6736(16)30069-1.
33. Reddel HK, FitzGerald JM, Bateman ED, et al. GINA 2019: a fundamental change in asthma management. Eur Respir J. 2019;53(6):1901046. doi:10.1183/13993003.01046-2019.
34. Bateman ED, Reddel HK, O'Byrne PM, et al. As-needed budesonide-formoterol versus maintenance budesonide in mild asthma. N Engl J Med. 2018;378(20):1877–1887. doi:10.1056/NEJMoa1715275.
35. ATS Committee on Proficiency Standards for Clinical Pulmonary Function Laboratories. ATS statement: guidelines for the six-minute walk test. Am J Respir Crit Care Med. 2002;166(1):111–117. doi:10.1164/ajrccm.166.1.at1102.
36. Smith RF, Johnson G, Ziesche S, et al. Functional capacity in heart failure: Comparison of methods for assessment. Circulation. 1993;87(6 Suppl):VI88–93. PMID:8500245.
37. Januzzi JL Jr, Camargo CA, Anwaruddin S, et al. The PRIDE study: NT-proBNP investigation of dyspnea in the emergency department. Am J Cardiol. 2005;95(8):948–954. doi:10.1016/j.amjcard.2004.12.032.
38. Maisel AS, Krishnaswamy P, Nowak RM, et al. Rapid measurement of BNP in the emergency diagnosis of heart failure. N Engl J Med. 2002;347(3):161–167. doi:10.1056/NEJMoa020233.
39. Dunlay SM, Roger VL, Weston SA, et al. Longitudinal changes in ejection fraction in heart failure patients with preserved and reduced EF. Circ Heart Fail. 2012;5(6):720–726. doi:10.1161/CIRCHEARTFAILURE.111.966366.
40. Pitt B, Williams G, Remme W, et al. The EPHESUS trial: eplerenone reduces mortality 30 days after randomization following acute myocardial infarction in patients with left ventricular systolic dysfunction and heart failure. J Am Coll Cardiol. 2005;46(3):425–431. This prespecified early-treatment analysis is included as a companion to the primary EPHESUS report.