

Original Contributions

Worsening Emergency Department Heart Rate Trajectory and Clinically Significant 7-Day Bouncebacks

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Abstract—Background: Discharge vital signs are used in emergency department (ED) risk assessment, but the association between heart rate (HR) trajectory and adverse outcomes after ED discharge remains unclear. **Study Objective:** To evaluate whether an increase in heart rate from triage to discharge is associated with clinically significant 7-day bounceback among discharged adult ED patients. **Methods:** We conducted a retrospective cohort study of adults discharged from a tertiary academic ED between February 1 and April 30, 2025. The primary predictor was HR increase ≥ 10 bpm from triage to discharge. The primary outcome was clinically significant 7-day bounceback, defined as a related return visit resulting in admission, observation, transfer, or death. Multivariable logistic regression adjusted for age, sex, comorbidities, and triage acuity. **Results:** The cohort included 6635 encounters. Any 7-day ED returns occurred at similar rates among patients with and without HR increase ≥ 10 bpm, 44 of 415 (10.6%) vs. 664 of 6220 (10.7%), respectively. In contrast, clinically significant bouncebacks occurred in 7 of 415 with HR increase ≥ 10 bpm (1.7%) compared with 30 of 6220 without it (0.5%). HR increase ≥ 10 bpm was associated with clinically significant bouncebacks after adjustment, aOR 3.10, 95% CI 1.35–7.13, $p = 0.008$. Discharge tachycardia alone was not associated with clinically significant bouncebacks, aOR 1.08, 95% CI 0.26–4.51, $p = 0.921$. **Conclusions:** Among discharged adult ED patients, HR increase ≥ 10 bpm was associated with clinically significant 7-day bounceback but not with any all-cause 7-day ED return. Heart rate trajectory may serve as an additional prompt for reassessment before ED

discharge. © 2026 Elsevier Inc. All rights are reserved, including those for text and data mining, AI training, and similar technologies.

Keywords—heart rate; tachycardia; vital signs; patient discharge; patient readmission

Introduction

Vital signs are a central component of emergency department (ED) risk assessment and disposition decision making. Among these, heart rate is particularly important because tachycardia may reflect physiologic stress, treatment response, medication effects, or evolving illness (1–4). Tachycardia is also incorporated into common emergency care frameworks, including sepsis screening, pulmonary embolism risk assessment, and shock evaluation, reinforcing its role as a marker of acuity and potential clinical instability (4–6).

Although abnormal vital signs at triage guide initial prioritization, evaluation, and treatment, their interpretation at the time of ED discharge is less clearly defined (6,7). Patients may remain tachycardic at discharge for many reasons, including fever, dehydration, medication exposure, withdrawal, or incomplete response to treatment. At the same time, persistent or worsening tachycardia may raise concern for unresolved illness or evolving clinical deterioration (2,6,8,9).

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Prior studies have shown that abnormal discharge vital signs are associated with adverse outcomes after ED discharge, including return visits, hospital admission, intensive care unit (ICU) admission, and death (4–6). Gabayan et al. found that a last-recorded ED heart rate ≥ 101 bpm was associated with approximately twice the odds of admission after discharge, odds ratio (OR) 2.00, 95% confidence interval (CI) 1.75–2.29 (5). More recent work has emphasized the importance of recognizing and addressing abnormal discharge vital signs before disposition, with proposed approaches including repeat vital sign assessment, provider acknowledgment, discharge checklists, and electronic health record alerts (4,6,10,11). However, isolated vital sign abnormalities have limited predictive value, and most existing work has evaluated discharge heart rate as a single absolute value (4–6,12).

Less is known about whether heart rate change during an ED visit adds clinical context beyond discharge heart rate alone. For example, a discharge heart rate of 105 bpm may have different meaning if it improved from 140 bpm than if it increased from 70 bpm. Prior work suggests that repeated vital sign measurements may better identify clinical deterioration than a single ED arrival measurement (12).

To address this gap, we evaluated whether worsening heart rate trajectory from triage to discharge was associated with clinically significant 7-day bouncebacks among adult ED patients discharged home. We hypothesized that patients with a worsening heart rate trajectory would have higher risk of clinically significant 7-day bouncebacks, and that heart rate trajectory would add clinical context beyond discharge tachycardia alone.

Materials and Methods

Study Design and Setting

We conducted a retrospective cohort study of adult patients discharged from a tertiary academic emergency department with approximately 55,000 annual visits. The study included ED encounters from February 1, 2025, through April 30, 2025. The study was reviewed and approved by the Health Sciences Institutional Review Board with a waiver of informed consent.

Study Population

Adult patients aged 18 years or older were eligible for inclusion if they were evaluated in the emergency department during the study period and discharged from the ED. For the primary analysis, patients were required to have

both a triage heart rate and a discharge heart rate documented in the electronic health record.

Patients were excluded if, during the index visit, they were admitted to the hospital, transferred to another facility, left without being seen, left against medical advice, eloped, were discharged to hospice or comfort-focused care, were pregnant, or had missing discharge heart rate data.

Predictor Variables

The primary predictor variable was worsening heart rate (HR) trajectory, defined as an increase in heart rate of at least 10 beats per minute from triage to discharge. Delta heart rate was calculated as discharge heart rate minus triage heart rate. Patients were categorized as having an HR increase ≥ 10 bpm if the calculated difference was greater than or equal to 10 bpm.

A secondary heart rate variable was discharge tachycardia, defined as discharge heart rate ≥ 100 bpm. This was evaluated separately to determine whether the absolute discharge heart rate was associated with clinically significant bouncebacks (CSBs) using the same adjusted modeling approach.

Outcome

The primary outcome was a clinically significant 7-day bounceback, defined as an unplanned return visit to the ED within 7 days of discharge for the same or related complaint or diagnosis that resulted in hospital admission, observation, transfer, or death. Any 7-day return visit, regardless of disposition, was evaluated as a secondary outcome.

Return visits within 7 days were identified electronically within the health system. Two emergency physicians reviewed return visits to determine whether they were for the same or a related complaint or diagnosis as the index ED encounter. A return was classified as a CSB if it was related and resulted in admission, observation, transfer, or death. Disagreements regarding relatedness were resolved by consensus. Reviewers agreed on classification for all reviewed cases, and a formal interrater reliability statistic was not calculated.

Data Collection and Covariates

Data were extracted from the electronic health record using automated reporting and manual chart review. Collected and calculated variables included patient demographics, age, sex, race, triage acuity, triage heart rate, discharge heart rate, Charlson Comorbidity Index, other vital signs, number of ED visits in the prior 30 days,

discharge diagnosis, and return visit diagnosis when applicable.

Additional variables that could plausibly influence heart rate trajectory or return risk were collected for descriptive and exploratory analyses. These included home beta blocker use, home calcium channel blocker use, ED crystalloid administration, ED bronchodilator use, ED analgesic use, stimulant use, cocaine use, methamphetamine use, and alcohol use.

Statistical Analysis

Descriptive statistics were used to summarize the study cohort. Continuous variables were reported as means with standard deviations or medians with interquartile ranges, as appropriate based on distribution. Categorical variables were reported as counts and percentages.

Patients were grouped according to whether they had an HR increase ≥ 10 bpm from triage to discharge. Baseline characteristics were compared between patients with and without HR increase ≥ 10 bpm. Categorical variables were compared using chi-square testing or Fisher's exact testing. Continuous variables were compared using t-tests for approximately normally distributed variables and Mann-Whitney U tests for skewed variables.

The primary outcome was a clinically significant 7-day bounceback, defined as a return visit for the same or related complaint or diagnosis resulting in admission, observation, transfer, or death. The proportion of patients with CSBs was compared between patients with and without HR increase ≥ 10 bpm. Fisher's exact test was used because of the low number of outcome events. Relative risks with 95% confidence intervals were calculated for unadjusted comparisons of 7-day return outcomes. Any 7-day return was evaluated as a secondary outcome. Timing of return was summarized by day of return for both any 7-day return and CSBs.

Exploratory analyses evaluated additional HR increase thresholds of ≥ 5 , ≥ 15 , and ≥ 20 bpm. For each threshold, sensitivity, specificity, positive likelihood ratio, negative likelihood ratio, and relative risk were calculated for CSB. Discharge tachycardia, defined as discharge HR ≥ 100 bpm, was evaluated using the same test characteristic approach. Delta HR was also evaluated as a continuous measure using receiver operating characteristic curve analysis, with area under the curve and 95% confidence interval reported.

Multivariable logistic regression was used to evaluate the association between HR increase ≥ 10 bpm and CSB after adjustment for selected baseline covariates. The adjusted model included HR increase ≥ 10 bpm, age, sex, Charlson Comorbidity Index, and triage acuity. Covariates were selected a priori based on clinical relevance, availability in the dataset, and the need to limit model

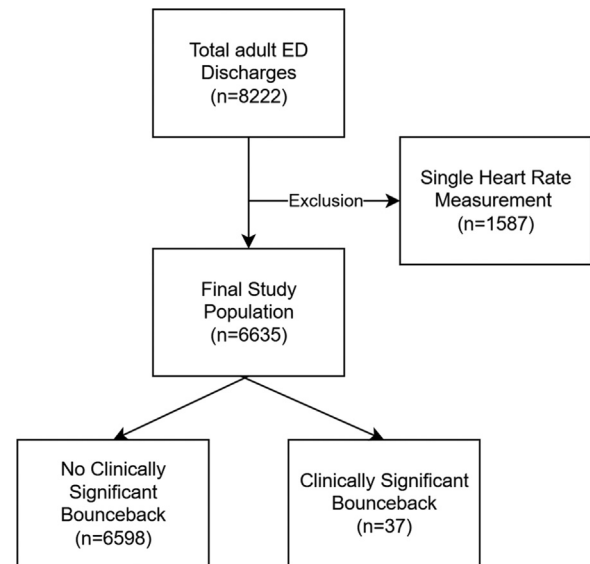


Figure 1. Study cohort flow diagram. ED = emergency department.

complexity because CSBs were rare. Adjusted odds ratios with 95% confidence intervals and *p* values were reported. The model was intended to assess whether the association between HR increase and CSB persisted after adjustment for measured covariates, rather than to establish a causal relationship.

A separate multivariable logistic regression model was used to evaluate the association between discharge tachycardia and CSB using the same covariates.

A sensitivity analysis was performed excluding patients who received ED medications with potential chronotropic effects, including bronchodilators, beta blockers, or calcium channel blockers during the ED encounter.

All analyses were performed using IBM SPSS Statistics. Statistical significance was defined as a two-sided *p* value < 0.05 .

Results

Study Cohort

During the study period, 8222 discharged adult ED encounters met initial eligibility criteria (Figure 1). Of these, 1587 encounters were excluded because they had only one documented heart rate and therefore could not be included in the heart rate trajectory analysis. The final analytic cohort included 6635 encounters with both triage and discharge heart rates documented. Among these, 37 had a CSB and 6598 did not.

Table 1. Baseline Characteristics of Discharged ED Patients by Heart Rate Increase ≥ 10 bpm.

Variable	No HR Increase ≥ 10 bpm <i>N</i> = 6220	HR Increase ≥ 10 bpm <i>N</i> = 415	<i>p</i> -Value
Age, mean $\pm$ SD, yr	43.2 $\pm$ 18.6	46.0 $\pm$ 21.4	0.003
Sex, n (%)			0.287
Male	3122 (50.2)	220 (53.0)	
Female	3098 (49.8)	195 (47.0)	
Race, n (%)			0.221
White	2854 (45.9)	189 (45.5)	
Black or African American	1480 (23.8)	114 (27.5)	
Asian	994 (16.0)	54 (13.0)	
Other	892 (14.3)	58 (14.0)	
Charlson Comorbidity Index, n (%)			0.023
0	3229 (51.9)	203 (48.9)	
1	1097 (17.6)	64 (15.4)	
2	796 (12.8)	47 (11.3)	
3	432 (6.9)	33 (8.0)	
4	329 (5.3)	35 (8.4)	
5	174 (2.8)	20 (4.8)	
6	81 (1.3)	7 (1.7)	
7+	82 (1.3)	6 (1.4)	
Triage acuity, n (%)			<0.001
2	668 (10.7)	60 (14.5)	
3	3740 (60.1)	271 (65.3)	
4	1761 (28.3)	82 (19.8)	
5	51 (0.8)	2 (0.5)	

ED = emergency department; HR = heart rate.

Data are presented as mean (SD) or n (%).

p-values were calculated using T-tests for continuous and chi-squared tests for categorical variables.

Baseline Characteristics

Baseline characteristics are shown in [Table 1](#). Patients with HR increase ≥ 10 bpm were slightly older than those without HR increase ≥ 10 bpm, 46.0 vs. 43.2 years, respectively ($p = 0.003$). Sex and race distributions were similar between groups ($p = 0.287$ and $p = 0.221$, respectively). Patients with HR increase ≥ 10 bpm had slightly higher comorbidity burden, with Charlson Comorbidity Index (CCI) ≥ 3 present in 101 of 415 patients (24.3%) compared with 1098 of 6220 patients (17.7%) without HR increase ≥ 10 bpm. Triage acuity also differed between groups ($p < 0.001$), with patients in the HR increase ≥ 10 bpm group more often triaged as acuity level 2 or 3 and less often as acuity level 4 or 5.

Additional clinical and treatment-related variables, including home rate-controlling medications, ED crystal-

loid administration, ED bronchodilator administration, ED analgesic provision, ED provision of betablockers or calcium channel blockers, and patient stimulant use, cocaine use, methamphetamine use, and alcohol use, were all evaluated and did not demonstrate a clinically meaningful association that warranted inclusion in the final adjusted model.

CSBs and All 7-Day Returns

Overall, 708 of 6635 encounters had an all-cause ED return within 7 days (10.7%). The rate of any 7-day return was similar among patients with HR increase ≥ 10 bpm and those without HR increase ≥ 10 bpm, occurring in 44 of 415 patients (10.6%) compared with 664 of 6220 patients (10.7%), respectively (RR 0.99, 95% CI 0.74–1.33, $p = 0.963$) ([Table 2](#)).

Table 2. Bouncebacks by Heart Rate Increase ≥ 10 bpm.

Variable	No HR Increase ≥ 10 bpm <i>N</i> = 6220	HR Increase ≥ 10 bpm <i>N</i> = 415	RR	95% CI	<i>p</i> -Value
Any 7-day return	664 (10.7)	44 (10.6)	0.99	0.74–1.33	0.963
Clinically Significant Bounceback, <i>n</i> (%)	30 (0.5)	7 (1.7)	3.5	1.55 – 7.92	0.007

RR = relative risk; CI = confidence interval; HR = heart rate; bpm = beats per minute.

Data are presented as *n* (%).

p-values were calculated using Fisher's exact test.

Among the 708 all-cause 7-day ED returns, 37.7% occurred by day 1, 67.1% by day 3, and 84.3% by day 5. The largest proportion of returns occurred on day 1 (26.0%), followed by day 2 (16.1%) and day 3 (13.3%).

In contrast, CSB occurred in 37 of 6635 encounters (0.6%). CSBs occurred in 7 of 415 patients with HR increase ≥ 10 bpm (1.7%) compared with 30 of 6220 patients without HR increase ≥ 10 bpm (0.5%) (Table 2). HR increase ≥ 10 bpm was associated with higher risk of CSB on unadjusted analysis (RR 3.50, 95% CI 1.55–7.92, *p* = 0.007).

CSB rates varied by triage acuity, occurring in 2 of 728 acuity level 2 encounters (0.3%), 32 of 4011 acuity level 3 encounters (0.8%), 3 of 1843 acuity level 4 encounters (0.2%), and 0 of 53 acuity level 5 encounters. The overall distribution differed across acuity categories (*p* = 0.013), although there was no significant linear trend by acuity level (*p* = 0.123).

Among the 37 CSBs, 2 occurred on the same day, 9 on day 1, 11 on day 2, 6 on day 3, 1 on day 4, 5 on day 5, 2 on day 6, and 1 on day 7. Overall, 28 of 37 CSBs (75.7%) had already occurred by day 3.

As defined by this study, all CSBs resulted in hospital admission. Thirty-three patients were admitted to a general floor while 4 were admitted to the ICU. No deaths occurred within 30 days of admission. The median hospital length of stay after admission was 2 days (IQR 1–4, range 1–36 days).

Bounceback diagnoses were most commonly infectious (18 patients, 48.6%), followed by cardiac or heart failure related diagnoses (7 patients, 18.9%), pain-related diagnoses (5 patients, 13.5%), hypovolemia (3 patients, 8.2%), gastrointestinal diagnoses (2 patients, 5.4%), alcohol withdrawal (1 patient, 2.7%), and substance intoxication (1 patients, 2.7%).

Exploratory Test Characteristics

In exploratory analyses, HR increase ≥ 10 bpm had low sensitivity but high specificity for a CSB (Table 3). Sensitivity was 18.9%, specificity was 93.8%, the positive likelihood ratio was 3.06, and the negative likelihood ratio was 0.86. The risk of CSB was 1.7% among patients with HR increase ≥ 10 bpm compared with 0.5% among those without HR increase ≥ 10 bpm, corresponding to a relative risk of 3.50. Across additional HR increase thresholds, higher cutoffs generally increased specificity but reduced sensitivity. Delta HR as a continuous measure had limited overall discrimination, with an area under the curve (AUC) of 0.576, 95% CI 0.471–0.682, *p* = 0.157. Discharge tachycardia had sensitivity of 5.4%, specificity of 95.0%, positive likelihood ratio of 1.08, and negative likelihood ratio of 1.00.

Multivariable Analysis

In multivariable logistic regression adjusting for age, sex, Charlson Comorbidity Index, and triage acuity, HR increase ≥ 10 bpm remained associated with CSB after adjustment (Table 4). Patients with HR increase ≥ 10 bpm had approximately threefold higher adjusted odds of CSB compared with patients without HR increase ≥ 10 bpm (aOR 3.10, 95% CI 1.35 to 7.13, *p* = 0.008). No other covariates in the adjusted model were significantly associated with CSB.

In a separate model using discharge tachycardia rather than HR increase ≥ 10 bpm as the primary heart rate variable, discharge tachycardia was not associated with CSB after adjustment (aOR 1.08, 95% CI 0.26–4.51, *p* = 0.921).

Table 3. Test Characteristics of Heart Rate Variables for Clinically Significant 7-Day Bounceback.

Heart Rate Increase	CSB if Positive	RR	Sensitivity	Specificity	LR+	LR-
HR increase ≥ 5 bpm	10/818, 1.2%	2.63	27.0%	87.8%	2.21	0.83
HR increase ≥ 10 bpm	7/415, 1.7%	3.50	18.9%	93.8%	3.06	0.86
HR increase ≥ 15 bpm	4/201, 2.0%	3.88	10.8%	97.0%	3.62	0.92
HR increase ≥ 20 bpm	1/98, 1.0%	1.85	2.7%	98.5%	1.84	0.99
Discharge tachycardia ≥ 100 bpm	2/331, 0.6%	1.09	5.4%	95.0%	1.08	1.00

CSB = clinically significant bounceback; HR = heart rate; bpm = beats per minute; RR = relative risk; LR+ = positive likelihood ratio; LR- = negative likelihood ratio.

Positive indicates the listed heart rate increase was present.

Table 4. Multivariable Logistic Regression Models for Clinically Significant 7-Day Bounceback.

Variable	Model 1, HR increase ≥ 10 bpm aOR	95% CI	<i>p</i> -Value	Model 2, Discharge Tachycardia aOR	95% CI	<i>p</i> -Value
Primary HR variable	3.096	1.346–7.126	0.008	1.076	0.256–4.512	0.921
Age (per year)	1.008	0.982–1.035	0.551	1.009	0.983–1.036	0.493
Sex (female vs. male)	0.946	0.498–1.798	0.866	0.941	0.495–1.786	0.852
CCI (per point)	1.095	0.844–1.422	0.494	1.096	0.847–1.418	0.487
Triage acuity	0.743	0.437–1.264	0.273	0.716	0.422–1.214	0.215

HR = heart rate; bpm = beats per minute; CCI = Charlson Comorbidity Index; CI = confidence interval; aOR = adjusted odds ratio.

Model 1 used HR increase ≥ 10 bpm as the primary heart rate variable. Model 2 used discharge tachycardia, defined as discharge HR ≥ 100 bpm, as the primary heart rate variable. Both models adjusted for age, sex, Charlson Comorbidity Index, and triage acuity.

Sensitivity Analysis

In a sensitivity analysis excluding patients who received ED medications with potential chronotropic effects, including bronchodilators, beta blockers, or calcium channel blockers, HR increase ≥ 10 bpm remained associated with CSB. CSB occurred in 6 of 391 patients with HR increase ≥ 10 bpm compared with 29 of 5922 patients without HR increase ≥ 10 bpm, 1.5% vs. 0.5%, respectively, RR 3.13, 95% CI 1.31–7.51, $p = 0.019$.

Discussion

In this retrospective cohort study of discharged adult ED patients, an increase in heart rate of at least 10 bpm from triage to discharge was associated with clinically significant 7-day bouncebacks after adjustment for age, sex, comorbidities, and triage acuity. In contrast, HR increase ≥ 10 bpm was not associated with any 7-day ED return, a broader outcome that included all return visits regardless of relatedness, disposition, or clinical significance.

Discharge tachycardia as a single value was not associated with CSBs. These findings suggest that heart rate trajectory may be more relevant to return visits requiring escalation of care than to ED returns in general and may provide additional clinical context beyond a single discharge heart rate threshold.

This distinction is important because emergency clinicians rarely interpret discharge vital signs in isolation. A discharge heart rate of 105 bpm may have different meaning if it improved from 140 bpm than if it increased from 70 bpm. Prior studies have shown that abnormal discharge vital signs are associated with short-term adverse outcomes, but isolated abnormalities are imperfect and should be interpreted in clinical context (5,6,12). Our findings build on this concept by suggesting that heart rate trajectory may add context to the final discharge heart rate and prompt reassessment before disposition.

The association observed in this study should not be interpreted as evidence that HR increase itself causes clinically significant return visits. Rather, a rising heart rate may reflect unresolved or evolving clinical factors not fully captured in structured data, including fever, hypovolemia, infection, withdrawal, medication effects, cardiopulmonary stress, or incomplete response to treatment. These factors may contribute to both heart rate change and the likelihood of return admission, observation, or transfer. Because tachycardia is a nonspecific physiologic finding with multiple possible causes, HR trajectory is best understood as an additional clinical data point rather than as a stand-alone determinant of disposition (1–3).

The exploratory test characteristic analyses support this cautious interpretation. HR increase ≥ 10 bpm had low sensitivity but high specificity for CSB, and delta HR as a continuous measure had limited overall discrimination on receiver operating characteristic curve (ROC) analysis. These findings indicate that HR trajectory should not be used as a screening test or rule-out tool. However, when present, a meaningful HR increase may still have practical value as a prompt for clinical reassessment. Although the event rate was low, the outcome was clinically meaningful because CSBs represented related return visits requiring escalation of care, including hospital admission and ICU admission. The potential value of HR trajectory therefore lies not in identifying a large number of events, but in providing a readily available signal associated with a higher-risk subset of ED returns.

The finding that HR increase was associated with CSBs but not with any 7-day returns also suggests that the signal may be more relevant to severity of return than to return visits in general. The sensitivity analysis excluding ED medications with potential chronotropic effects showed a similar association, suggesting that the observed relationship was not explained solely by these medication exposures.

The lack of association between discharge tachycardia and CSB should be interpreted cautiously and does not suggest that discharge tachycardia is unimportant. Prior work has shown that discharge tachycardia may be associated with short term adverse outcomes after ED discharge and should be interpreted in clinical context (5,6,12). One possible explanation is that discharge tachycardia is a familiar and visible abnormality, prompting additional reassessment, treatment, observation, or reconsideration of disposition before discharge. Patients ultimately discharged with tachycardia may therefore represent a selected subgroup in whom clinicians had already considered the abnormal heart rate and determined that discharge was appropriate. We did not review whether discharge tachycardia was explicitly addressed in clinician medical decision making, which limits our ability to evaluate this possibility.

The observed difference between discharge tachycardia and heart rate trajectory may also reflect the limitations of a single binary threshold. Patients with markedly elevated triage heart rates may improve yet remain mildly tachycardic at discharge, whereas patients with normal initial heart rates may have more opportunity to demonstrate a meaningful increase. The discharge tachycardia analysis may also have been underpowered because CSBs were rare. These findings suggest that heart rate trajectory may add clinical context by helping distinguish whether the final heart rate reflects improvement, stability, or worsening during the ED encounter.

This study also extends prior work on repeated vital sign assessment during care. Quinten et al. prospectively evaluated 359 ED patients. In that study, admission heart rate was associated with deterioration, OR 1.01, while changes over time in mean arterial pressure, OR 1.04, and respiratory rate, OR 1.44, were also associated with deterioration. Repeated measurements of heart rate and body temperature were not associated with deterioration (13). Although that study focused on admitted patients with suspected infection or sepsis and used a different outcome than ours, it supports the broader concept that vital sign trends during ED care may add information beyond a single measurement. Our study applies this concept to discharged ED patients by evaluating whether heart rate change from triage to discharge is associated with clinically significant 7-day bouncebacks.

The distribution of CSB diagnoses supports the clinical relevance of considering heart rate trajectory during discharge review. Nearly half of CSBs were infectious, followed by cardiac or heart failure related diagnoses, pain-related diagnoses, hypovolemia, withdrawal or toxicologic diagnoses, and gastrointestinal diagnoses. These categories are consistent with conditions in which a rising heart rate may reflect ongoing physiologic stress, incomplete response to treatment, or evolving illness. Similar

mechanisms have been described in prior work evaluating vital signs, tachycardia, infection, cardiovascular disease, medication effects, and physiologic instability (2,3,6,8). However, because the number of CSB events was small, these diagnostic patterns should be interpreted descriptively.

Moreover, broader cardiovascular literature also supports the concept that heart rate is a dynamic physiologic measure rather than a static value. Resting heart rate and changes in heart rate over time have been associated with cardiovascular events, heart failure hospitalization, myocardial infarction, stroke, mortality, and lifespan in populations outside the ED (14–19). Although these studies were conducted in different clinical settings, they support the biologic plausibility that changes in heart rate may reflect changes in physiologic stress or clinical status over time.

ED discharge decisions also reflect distributed decision making across time, team members, and documentation. Triage assessment, nursing reassessment, clinician evaluation, treatment response, repeat vital signs, and discharge planning are integrated before disposition (20). In this context, HR trajectory may help connect information gathered early in the ED encounter with the final discharge assessment.

These findings have practical implications for ED discharge decision making. Because HR trajectory is derived from routinely collected vital signs, an increase of at least 10 bpm from triage to discharge can be incorporated into discharge review as a practical signal for reassessing the patient's clinical status and discharge plan. This approach is consistent with recommendations that abnormal discharge vital signs be acknowledged and interpreted in clinical context rather than treated as isolated thresholds (6,11). Therefore, we recommend that heart rate trajectory be considered as one additional data point within the broader discharge assessment.

Limitations

This study has several limitations. First, it was retrospective and conducted at a single academic ED, which may limit generalizability. This design also depends on the accuracy and completeness of structured electronic health record (EHR) data and clinical documentation. Second, CSBs were rare, with 37 events (0.6%). This limited statistical precision and required an adjusted model with selected clinically relevant covariates. Although the model adjusted for measured baseline factors, residual confounding remains likely. Third, the study could not fully determine why a patient's heart rate increased. Potential contributors such as fever, dehydration, medication exposure, withdrawal, treatment timing, and evolving illness may have influenced both heart rate trajectory and

bounceback risk. Fourth, heart rate trajectory was calculated using triage and discharge heart rates only. Intermediate vital signs, timing of measurements, and response to specific ED interventions were not fully captured. Fifth, relatedness of return visits to the index visit was reviewed by two emergency physicians using a standardized definition, but reviewers were not formally blinded to HR trajectory, no pilot calibration sample was performed, and formal interrater reliability was not calculated because reviewers agreed on all reviewed cases. Reviewers did not subjectively determine whether a bounceback was clinically significant. Clinical significance was assigned using prespecified disposition-based criteria, including admission, observation, transfer, or death. Misclassification of relatedness remains possible. Sixth, return visits were identified within the available records, so returns to other health systems outside of our medical records may have been missed. Finally, this study identifies an association and cannot establish that interventions based on HR trajectory would reduce adverse outcomes. The study also could not determine whether any patient should have been admitted during the index ED encounter or whether admission would have prevented the bounceback. Future studies with larger cohorts, more detailed repeated vital sign data, and external validation are needed to better define how heart rate trajectory should be incorporated into ED discharge workflows.

Conclusion

An increase in heart rate of at least 10 bpm from triage to discharge was associated with clinically significant 7-day bounceback, but not with any 7-day ED return. Discharge tachycardia alone was not associated with CSB. Heart rate trajectory may provide additional clinical context beyond the absolute discharge heart rate and may serve as a practical signal for reassessment before ED discharge. Further validation in larger, multicenter cohorts is needed before this measure is incorporated into formal discharge decision tools.

IRB Statement

This study was reviewed and approved by the Tufts Health Sciences Institutional Review Board with a waiver of informed consent.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this manuscript.

CRediT authorship contribution statement

Sami Shayya: Writing – original draft, Visualization, Supervision, Software, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Cornelis Hollenbach:** Data curation. **Eric Schaar:** Data curation. **Richard Chu:** Data curation. **Sabri Bilse:** Data curation. **Kristian Najdoski:** Data curation. **Dailyn Ortega Gonzalez:** Data curation. **Mary Meinke:** Methodology, Data curation.

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Article Summary

1. Why is this topic important?

Discharge vital signs are commonly used in emergency department disposition decisions, but most prior work has focused on the absolute discharge vital sign value. Less is known about whether heart rate change during the ED encounter adds useful clinical context before discharge.

2. > What does this study attempt to show?

This study evaluated whether an increase in heart rate of at least 10 bpm from triage to discharge was associated with clinically significant 7-day bounceback among discharged adult ED patients. A clinically significant bounceback were defined as a related return visit resulting in admission, observation, transfer, or death.

3. What are the key findings?

An HR increase ≥ 10 bpm from triage to discharge was associated with approximately threefold higher adjusted odds of clinically significant 7-day bounceback. In contrast, HR increase ≥ 10 bpm was not associated with any all-cause 7-day ED return, and discharge tachycardia alone was not associated with clinically significant bouncebacks in a separate adjusted model. Heart rate trajectory may add clinical context beyond the absolute discharge heart rate.

4. How is patient care impacted?

Heart rate trajectory may serve as one additional data point during ED discharge assessment. A rising heart rate may prompt reassessment of fever, volume status, infection risk, cardiopulmonary symptoms, response to ED treatment, and discharge planning, but should not be used as a stand-alone disposition rule.