The association between systolic blood pressure and heart rate in emergency department patients: a multicenter cohort study

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Original Contributions

THE ASSOCIATION BETWEEN SYSTOLIC BLOOD PRESSURE AND HEART RATE IN EMERGENCY DEPARTMENT PATIENTS: A MULTICENTER COHORT STUDY

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□ **Abstract—Background: Guidelines and textbooks assert that tachycardia is an early and reliable sign of hypotension, and an increased heart rate (HR) is believed to be an early warning sign for the development of shock, although this response may change by aging, pain, and stress. Objective: To assess the unadjusted and adjusted associations between systolic blood pressure (SBP) and HR in emergency department (ED) patients of different age categories (18–50 years; 50–80 years; > 80 years). Methods: A multicenter cohort study using the Netherlands Emergency department Evaluation Database (NEED) including all ED patients ≥ 18 years from three hospitals in whom HR and SBP were registered at arrival to the ED. Findings were validated in a Danish cohort including ED patients. In addition, a separate cohort was used including ED patients with a suspected infection who were hospitalized from whom measurement of SBP and HR were available prior to, during, and after ED treatment. Associations between SBP and HR were visualized and quantified with scatterplots and regression coefficients (95% confidence interval [CI]). Results: A total of 81,750 ED patients were included from the NEED, and a total of 2358 patients with a suspected infection. No associations were found between SBP and HR in any age category (18–50 years: −0.03 beats/min/10 mm Hg, 95% CI −0.13–0.07, 51–80 years: −0.43 beats/min/10 mm Hg, 95% CI −0.38 to −0.50, > 80 years: −0.61 beats/min/10 mm Hg, 95% CI −0.53 to −0.71), nor in different subgroups of ED patient. No increase in HR existed with a decreasing SBP during ED treatment in ED patients with a suspected infection. Conclusion: No association between SBP and HR existed in ED patients of any age category, nor in ED patients who were hospitalized with a suspected infection, even during and after ED treatment. Emergency physicians may be misled by traditional concepts about HR disturbances because tachycardia may be absent in hypotension.**

□ **Keywords—hypotension; shock; critical care; emergency medical services; geriatric emergency medicine**

Ethical statement and consent to participate: The study was approved by the medical ethics committee of the Máxima Medical Centre (reference N20.092) and has been performed in accordance with the declaration of Helsinki. A consent to participate was waived due to the retrospective design.

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INTRODUCTION

Several textbooks and guidelines, such as Advanced Trauma Life Support and the Surviving Sepsis Campaign, assert that tachycardia is an essential part of the physiological response to hypotension (1–3). Consequently, tachycardia is often used as an early marker of hemodynamic instability (2,3). Nonetheless, in trauma and sepsis patients, hypotension is often not accompanied by tachycardia, and previous studies suggest not relying solely on heart rate (HR) disturbances to recognize a progressive fall in blood pressure (4–9).

Even if HR increases with decreasing blood pressure, many factors, such as pain or anxiety, may cause a tachycardic response and thus, HR may not be a reliable vital sign in an undifferentiated patient population, such as in the emergency department (ED). More importantly, the HR response in situations with lowering blood pressure may be blunted with higher age, possibly by a decreased chronotropic beta-adrenergic responsiveness (10,11). For this reason, we hypothesized that an association between systolic blood pressure (SBP) and HR may exist in younger, but is less clear or absent in older, ED patients. A better understanding of the association between SBP and HR helps to assess the usefulness of HR as a vital sign to detect hypotension in acutely ill patients.

This study aims to assess the association between SBP and HR in the ED setting in different age categories.

METHODS

Study Design and Setting

This multicenter cohort study used three separate cohorts: the Netherlands Emergency department Evaluation Database (NEED), a quality registry for Dutch Emergency Departments (www.stichting-NEED.nl), and the Danish Multicenter Cohort (DMC) to externally validate our findings, and a cohort including patients with a suspected infection (Sepsis op Spoedeisende Hulp [SOS] cohort). The NEED contained data from three EDs, a tertiary care center and two urban teaching hospitals (January 2017–September 2020). The DMC included all ED patients aged $\geq$ 18 years from two Level II EDs with data between 2008 and 2013. Patients were consecutively sampled in relation to previous prospective studies at two Level II emergency centers. This cohort was chosen for external validation to include an ED population similar to that in the NEED, but in a different country and period. The cohort has been described in detail previously (12,13). The SOS cohort included patients who were hospitalized from the ED with a suspected infection from two tertiary care centers and one urban teaching hospital in the Netherlands, with data between 2011 and 2013. This was a retrospective analysis of prospectively collected data. The cohort has been described in detail previously (14). This cohort enabled the exploration of vital signs within one patient during resuscitation, as it contained vital sign registrations at three different time points in the ED.

Patients were stratified by age categories (18–50, 51–80, $>$ 80 years). These age categories were chosen because the incidence of hypertension is low until the age of 50 years in the Netherlands, after which it gradually increases (www.cbs.nl). The cut-off at 80 years has been used in previous studies (13,15).

Participants

In the NEED and DMC, all consecutive patients $\geq$ 18 years presenting to the ED were included if both SBP and HR were registered.

The SOS cohort included consecutive ED patients aged 17 years and older—with suspected infection and an urgent to very urgent triage category according to the Manchester triage system—who were admitted to the hospital and treated with intravenous antibiotics. For the current study, patients aged 17 years and younger were excluded from the database.

Data Collection

Previously, we described in detail how vital signs were measured and which data were collected in the NEED (16). In summary, we collected demographic variables, disease severity, proxies for comorbidity and complexity, vital signs, laboratory results, and outcomes. One set of vital signs (HR, SBP, peripheral oxygen saturation, respiratory rate, temperature, diastolic blood pressure) was recorded per patient, measured at the beginning of ED presentation prior to ED treatment. Vital signs were not recorded if this was not necessary for the ED presentation. This was similar for the DMC. In the SOS, vital signs were registered prior to ED treatment (Time 0h at arrival identical to the NEED cohort), during ED treatment (approximately around time 30 min), and after ED treatment (prior to admission to the ward). During ED stay, the lowest SBP and the highest HR value were registered. Also, the use of beta-blocking agents and calcium antagonists was registered only in this cohort.

Data Analyses

Descriptive analyses

Data were presented as mean (standard deviation [SD]) if normally distributed and median (interquartile range) if skewed.

Main statistical analyses

Univariable associations between SBP and HR were investigated and visualized using scatterplots for three age categories. Both a linear and locally estimated scatterplot smoothing fit with 95% confidence intervals (CI) were fitted to the data. In the figures, the size of the dots indicates the precision of the estimate for observed HR and is based on the inverse of the standard deviation. The larger the dot, the higher the precision. A linear regression coefficient with 95% CI and an R-squared were also presented.

To correct the association between SBP and HR for other vital sign values and pain scores, an additional analysis was performed. We investigated the association between SBP and HR using the R package "mgcv" to fit a generalized additive regression model adjusted for other vital signs and pain scores, which were left constant at 'normal' values (respiratory rate 18 breaths/min, peripheral oxygen saturation 97%, temperature 36°C), numeric rating scale for pain 0), on the NEED data. Missing numeric rating scale was considered as a score of 0. If other vital signs were missing the patient was excluded. This analysis was intended to be descriptive, and therefore we did not attempt formal statistical inference in terms of *p*-values and confidence intervals.

In the SOS we assessed whether the use of beta-blocking agents or calcium antagonists affected our results by linear fits for patients with beta-blocking agents and patients without beta-blocking agents. A linear regression coefficient with a *p*-value and an R-squared was presented. In addition, we assessed whether deterioration of blood pressure was accompanied by an increasing HR. We therefore calculated the deltaSBP = SBP after ED treatment − SBP prior to ED treatment, and similarly, a deltaHR. We assessed the relationships between those two variables using a scatterplot with linear fit.

Subgroup analyses

We performed four subgroup analyses in the NEED. The first subgroup analysis included all patients with an SBP < 120 mm Hg, to put more focus on the hypotensive patients in this study. In this subgroup, we compared the in-hospital mortality rates in patients with a low HR response (below the median) to hypotension with a high HR response (above the median). In this way we could study whether the absence of a tachycardic response had a poor outcome compared with patients with a tachycardic response.

To assess whether the etiology of a low SBP and severity of illness affected the univariable association with HR, the relationship between SBP and HR were also investigated for the subgroups' suspected infection and trauma patients (potentially having hemorrhage) in the NEED, and for patients admitted to the intensive care unit (ICU). In the subgroups' suspected infection and trauma, the most likely cause for hypotension is septic shock and hemorrhagic shock, respectively, whereas other reasons are less likely. In this way, we could assess the association between SBP and HR in two types of shock patients. Patients with a suspected infection were selected in the NEED if blood cultures were obtained and if they were hospitalized. Trauma patients were selected if they were triaged as very urgent or immediate (according to the Manchester Triage System or Dutch Triage Standard) and by the following triage-presenting complaints: severe trauma; facial complaints; fall; extremity complaints; large-scale incidents; neck pain; thorax injury; head injury; wounds. Additionally, a radiological test had to be performed. Patients admitted to the ICU were registered in the NEED and were studied as the last subgroup.

All statistical analyses were performed in RStudio (packages "rms," "dplyr," and "mgcv").

RESULTS

Patient Characteristics

The NEED contained 148,828 patients aged $\geq$ 18 years. In total, 81,750 (54.9%) patients could be included in whom both SBP and HR were registered. See the flow diagram in Figure 1. The mean age of patients in the NEED was 61 years (SD 19). Mean SBP was higher in older patients aged $>$ 80 years, with 141 mm Hg (SD 34), compared with the youngest age category, with mean SBP of 124 mm Hg (SD 26). Patient characteristics are described in Table 1.

Main Results

Figure 2 demonstrates the association between SBP and HR in three age categories at arrival to the ED. No association between HR and SBP was found in any age group. No association between SBP and HR was found in any subgroup of hypotensive patients, trauma patients, patients with a suspected infection, and ED patients admitted to the ICU (Figure 3). Adjustment for other vital signs and pain scores did not affect the association between SBP and HR (Figure 4).

The SOS cohort included 2358 patients with a suspected infection. Patient characteristics are described in Supplementary Table 1 (available online). No clear increase in HR could be found, with decreasing SBP for any age category prior to, during, or after ED treatment (Figure 5). The use of beta-blocking agents or calcium antagonists did not affect the relationship. If the vital signs after ED treatment of an individual patient were compared with the vital signs prior to ED treatment, no

Table 1. Patient Characteristics from the Netherlands Emergency Department Evaluation Database (NEED)

NEED Cohort	18–50 Years (n = 22,617)	51–80 Years (n = 46,350)	> 80 Years (n = 12,783)	All (n = 81,750)
Age, years				
Mean (SD)	35 (9.8)	67 (8.4)	86 (3.9)	61 (19)
Sex				
Female	12,170 (53.8%)	21,054 (45.4%)	7144 (55.9%)	40,368 (49.4%)
Triage category, n (%)				
Immediate	1044 (4.6%)	3050 (6.6%)	788 (6.2%)	4882 (6.0%)
Very urgent	6534 (28.9%)	15,037 (32.4%)	3891 (30.4%)	25,462 (31.1%)
Urgent	9771 (43.2%)	19,349 (41.7%)	5362 (41.9%)	34,482 (42.2%)
Nonurgent	4885 (21.6%)	8181 (17.7%)	2516 (19.7%)	15,582 (19.1%)
Missing	383 (1.7%)	733 (1.6%)	226 (1.8%)	1342 (1.6%)
Top five presenting complaints, n (%)				
Feeling unwell	3004 (13.3%)	10,589 (22.8%)	3373 (26.4%)	16,966 (20.8%)
Abdominal pain	4909 (21.7%)	5346 (11.5%)	870 (6.8%)	11,125 (13.6%)
Dyspnea	1798 (7.9%)	6630 (14.3%)	2067 (16.2%)	10,495 (12.8%)
Chest pain	2481 (11.0%)	6540 (14.1%)	1136 (8.9%)	10,157 (12.4%)
Extremity complaints	1277 (5.6%)	3027 (6.5%)	1573 (12.3%)	5877 (7.2%)
Miscellaneous	8670 (38.3%)	13,379 (28.9%)	3515 (27.5%)	25,564 (31.3%)
Missing	478 (2.1%)	839 (1.8%)	249 (1.9%)	1566 (1.9%)
Systolic blood pressure (mm Hg)				
Mean (SD)	124 (26)	135 (32)	141 (34)	133 (31)
Heart rate (beats/min)				
Mean (SD)	87 (20)	86 (22)	83 (21)	86 (21)
Respiratory rate (breaths/min)				
Mean (SD)	17 (5)	18 (5)	19 (6)	18 (5)
Missing	6865 (30.4%)	9729 (21.0%)	2385 (18.7%)	18,979 (23.2%)
Peripheral oxygen saturation (%)				
Mean (SD)	98.4 (2.5)	96.7 (3.6)	96.0 (3.8)	97.1 (3.5)
Missing	823 (3.6%)	1819 (3.9%)	509 (4.0%)	3151 (3.9%)
Temperature (°C)				
Mean (SD)	37.1 (0.8)	37.0 (0.9)	36.9 (0.9)	37.0 (0.9)
Missing	4573 (20.2%)	7944 (17.1%)	2312 (18.1%)	14,829 (18.1%)
Numeric rating scale for pain (1 to 10)				
Median (IQR)	4 (1–6)	2 (0–5)	2 (0–4)	3 (0–5)
Missing	13,088 (57.9%)	30,002 (64.7%)	8698 (68.0%)	51,788 (63.3%)
Supplemental oxygen				
yes	429 (1.9%)	2879 (6.2%)	1353 (10.6%)	4661 (5.7%)
Missing	6086 (26.9%)	10,745 (23.2%)	2237 (17.5%)	19,068 (23.3%)
Fluid administration (mL)				
0 cc	11,407 (50.4%)	26,027 (56.2%)	7289 (57.0%)	44,723 (54.7%)
0–500 cc	2031 (9.0%)	4314 (9.3%)	1322 (10.3%)	7667 (9.4%)

(*continued on next page*)

Table 1. (*continued*)

NEED Cohort	18–50 Years (n = 22,617)	51–80 Years (n = 46,350)	> 80 Years (n = 12,783)	All (n = 81,750)
> 500 cc	2320 (10.3%)	5159 (11.1%)	1239 (9.7%)	8718 (10.7%)
Missing	6859 (30.3%)	10,850 (23.4%)	2933 (22.9%)	20,642 (25.3%)
In-hospital mortality, n (%)				
died	90 (0.4%)	1197 (2.6%)	770 (6.0%)	2057 (2.5%)
Missing	188 (0.8%)	688 (1.5%)	264 (2.1%)	1140 (1.4%)
ICU admission, n (%)				
ICU admission	350 (1.5%)	924 (2.0%)	140 (1.1%)	1414 (1.7%)
Missing	318 (1.4%)	397 (0.9%)	66 (0.5%)	781 (1.0%)

IQR = interquartile range; GCS = Glasgow Coma Scale; ICU = intensive care unit.

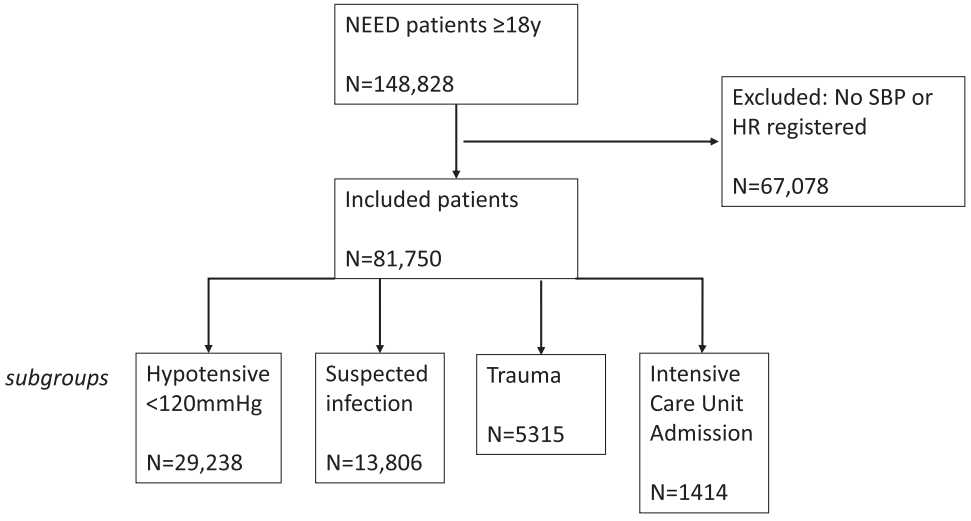

Figure 1. Flow diagram throughout the Netherlands Emergency department Evaluation Database (NEED) cohort. SBP = systolic blood pressure; HR = heart rate.

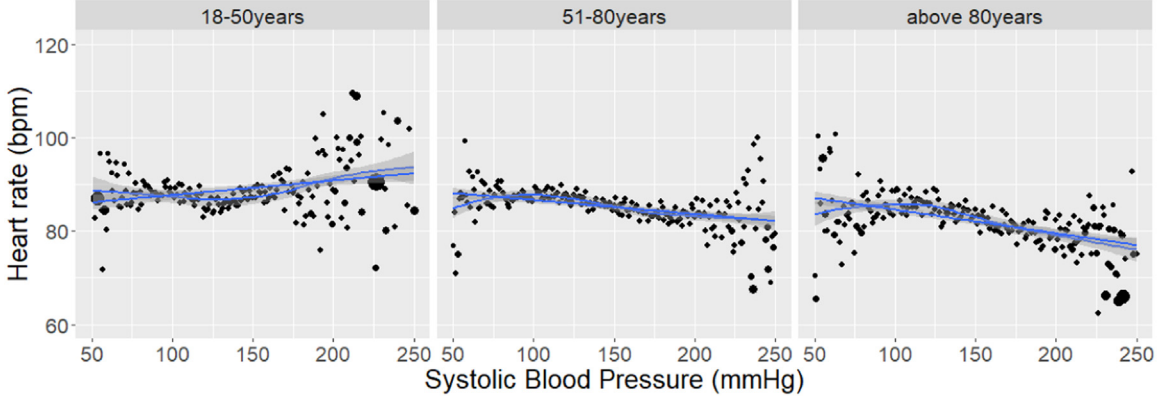

Figure 2. The univariable relationship between systolic blood pressure (SBP) and heart rate (HR) stratified by age categories in the Netherlands Emergency department Evaluation Database (NEED) cohort (n = 81,750). The size of the dots indicates the precision of the estimate for observed HR and is based on the inverse of the standard deviation. The larger the dot, the higher the precision. Both a linear and locally estimated scatterplot smoothing fit with 95% confidence intervals were fitted to the data. No association was found in any age category between SBP and HR.

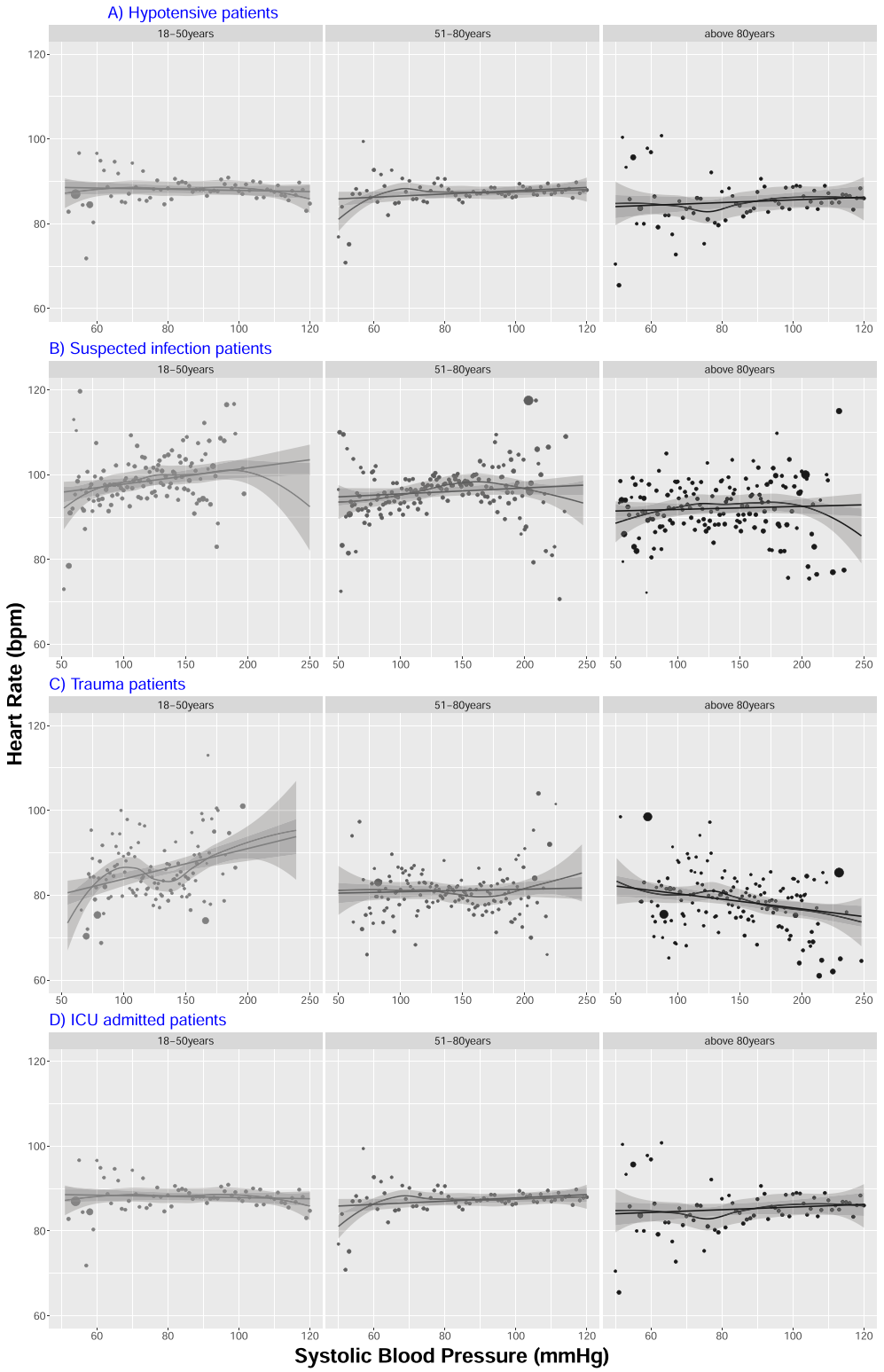

Figure 3. Subgroup analyses in the Netherlands Emergency department Evaluation Database (NEED) registry. Scatterplots are presented for the association between systolic blood pressure and heart rate in different age categories. Panel A shows the association between systolic blood pressure (SBP) and heart rate in a subgroup of patients with an SBP < 120 mm Hg (n = 29,238). Panel B shows a subgroup of patients with a suspected infection (n = 13,806). Panel C shows a subgroup of trauma patients (n = 5315). Panel D shows a subgroup of emergency department patients who were admitted to the Intensive Care Unit (ICU; n = 1414). No association was found in any age category between SBP and heart rate.

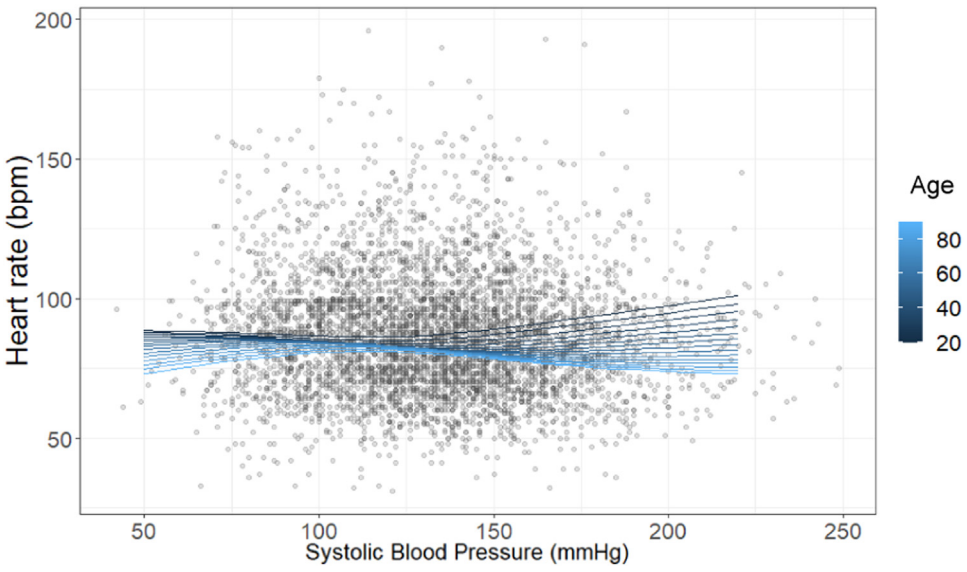

Figure 4. The associations between systolic blood pressure and heart rate and age is shown using a generalized additive logistic regression model, adjusted for temperature, peripheral oxygen saturation, respiratory rate, and a pain score (numeric rating scale 0 to 10). No association was found in any age between systolic blood pressure and heart rate.

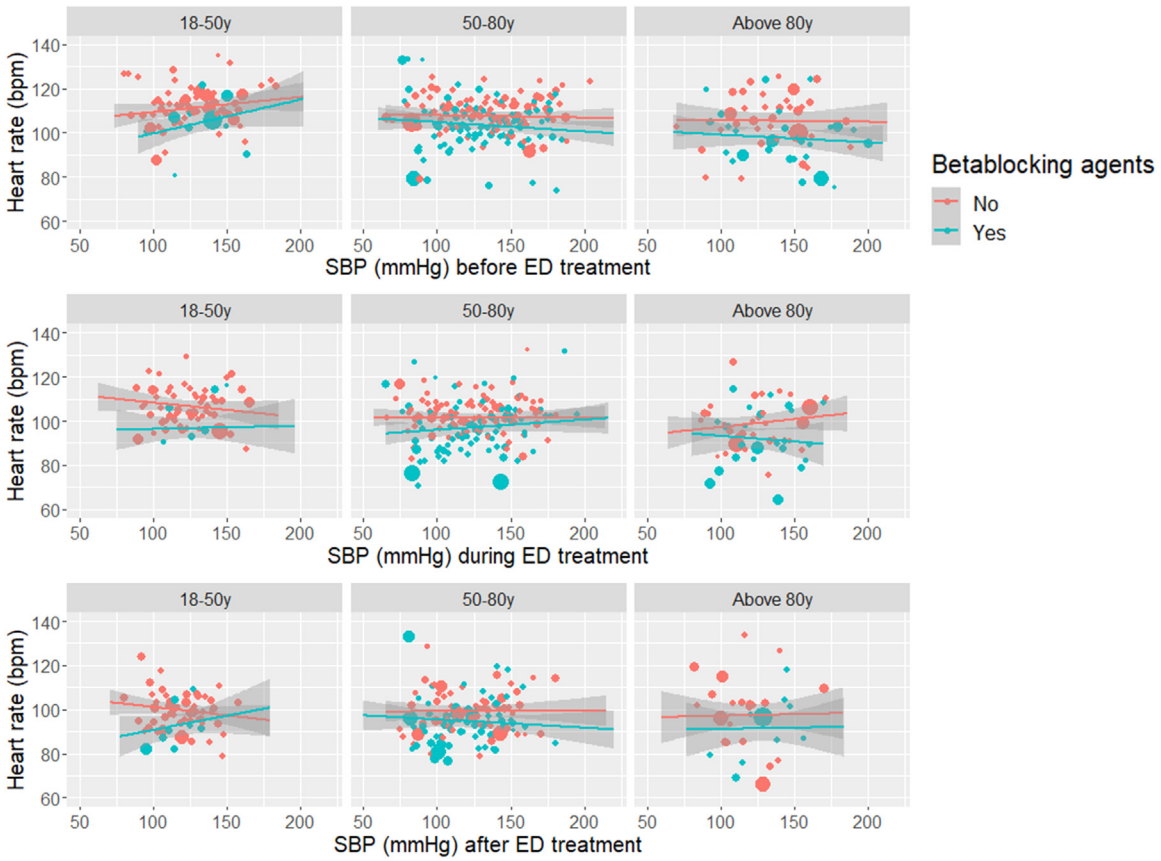

Figure 5. The univariable relationship between systolic blood pressure (SBP) and heart rate (HR) stratified by age categories and time of registration in a cohort of patients with a suspected infection (n = 2358). A linear line was fitted to the data. The relationship was assessed prior to emergency department (ED) treatment (T0), during ED treatment (T30min) and after ED treatment just prior to ward admission. Patients with beta-blocking agents or calcium antagonists were marked. No association was found in any age category between SBP and HR.

increase in deltaHR existed with a decreasing SBP during resuscitation in the ED (e.g., a negative deltaSBP; see Supplementary Figure 1, available online).

External validation in a Danish cohort of ED patients showed similar results (Supplementary Table 2, Supplementary Figure 2, available online). No clear increase in HR could be found with decreasing SBP for any age category.

Regression coefficients and R-squared for the different cohorts and subgroups are presented in Supplementary Table 3 (available online). Although some regression coefficients were statistically significant, they were close to zero and therefore not clinically relevant.

In the subgroup with hypotensive patients (SBP $<$ 120 mm Hg) we compared mortality rates of patients with a low HR and patients with a high HR response. Mortality was higher in the patients with a high HR (Supplementary Table 4, available online). However, the hypotensive patients aged $>$ 80 years still had a 7.3% mortality risk in the absence of a tachycardic response.

DISCUSSION

In this multicenter cohort study, no association between SBP and HR was found in ED patients of any age category or subgroup, including suspected infection, trauma, and ICU-admitted patients. In the ED, tachycardia alone may therefore not be helpful in detecting hypotension.

Although several textbooks and guidelines assert that tachycardia is an early and reliable sign of hypotension to maintain sufficient cardiac output and circulating volume, low blood pressure was not accompanied by an increase in HR in the present study (1–3,17). Textbooks are based on theoretic physiological principles explaining that compensated hypotension is characterized by intense sympathetic stimulation of the circulation elicited by baroreceptor reflexes (1,17,18). However, several animal studies have shown an absence of tachycardia in hemorrhage, which is in line with the findings of the present study (18–21). The absence of tachycardia as a response to hypotension during hemorrhage has been explained to be a vagal reflex (7,21–23).

In sepsis it is currently believed that inflammation leads to vasodilatation, which alters cardiac output due to preload reduction (2,24,25). As a result, sympathetic activation is triggered and should lead to a compensated tachycardia and vasoconstriction. Nevertheless, decreasing blood pressure was not accompanied by an increased HR in younger and older patients on average cohort level. This finding follows several studies that have demonstrated that an altered HR variability in sepsis is common and correlates with the severity of the systemic infection with a higher mortality rate (8,9).

At the start of this study we hypothesized that an association between SBP and HR could be absent in older patients due to the decreasing chronotropic response with increasing age (10,11). However, the association was also lacking in younger patients. Our results were confirmed in a Danish multicenter cohort, implying that they can be generalized to other ED settings and times. Also, we have shown that adjusting for other vital signs and pain did not affect the association between SBP and HR in ED patients. We can only speculate why an increased HR did not accompany hypotension in the ED. A potential explanation is the large variety in ED presentations. Although patients with deranged hemodynamics, such as septic patients, would be expected to have a combination of tachycardia and low blood pressure, other ED patients presenting with pain or anxiety disorders could have tachycardia with high blood pressure. Other factors such as changes in blood volume, pain, anxiety, parasympathetic and sympathetic stimuli, and circulating hormones are likely explanations that may play an essential role in ED patients. Due to this heterogeneity, no association between SBP and HR may have been found.

The NEED only contained vital sign registrations at arrival to the ED, but repeated analyses in a cohort with multiple registrations during and after ED treatment showed similar results. Remarkably, a decrease in SBP during and after resuscitation was not accompanied by an increase in HR in individual patients with a suspected infection.

The importance of our observations lies in the fact that hypotension cannot be recognized solely by the presence of tachycardia, and so HR may be an unreliable vital sign to screen for physiological derangement and hypotension in ED patients of all age categories, whereas guidelines such as Advanced Trauma Life Support continue to use HR as one of the vitals to classify the degree of hemorrhagic shock (1–3). In this study we demonstrated that mortality rates are lower in hypotensive patients without a tachycardic response than in patients with a tachycardia. Hypothetically, the patients with an SBP below 120 mm Hg and absent tachycardic response might not have essential hypotension, for example, a perfusion problem. Nonetheless, the in-hospital mortality rate was still substantially high in older patients, possibly caused by a decreased chronotropic beta-adrenergic responsiveness or by beta-blocking medication. Therefore, emergency physicians should not be misled by traditional concepts about HR disturbances (1–3).

Despite several strengths like the large sample size and the fact that we performed analyses in multiple independent cohorts, several limitations merit emphasis. First, only one set of vital signs at ED arrival was available in the NEED database, which may have affected our results. Possibly the first set of vital signs is affected by measurement artifacts, or many more confounders, such as mental

stress and pain affecting HR, which could blur the association with SBP. However, we assessed the relationship between SBP and HR in a cohort including patients with a suspected infection at three different times during ED stay (Supplementary Figure 1), which showed similar results. Secondly, we could not adjust analyses in the NEED or the ICU dataset for potential confounders such as anxiety or vasopressor/inotropic agents. Nonetheless, we have shown no clear relationship between SBP and HR exists, even after adjusting for vital signs and pain. Finally, we assessed the relationship between SBP and HR at cohort level. A decreasing SBP may still be accompanied by an increasing HR in individual patients, even though no increased HR was found, with a decreasing SBP in patients with suspected infection in the SOS cohort.

In summary, the present study indicates that an association between SBP and HR cannot be detected in ED patients of any age category or subgroup. Acute care guidelines should acknowledge that HR may not be a reliable vital sign to detect physiological derangement in the ED.

Declaration of Competing Interest

The authors declare that they have no competing interests.

SUPPLEMENTARY MATERIALS

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jemermed.2023.04.009.

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ARTICLE SUMMARY

1. Why is this topic important?

The compensatory response of heart rate (HR) to hypotension may be blunted in older emergency department (ED) patients due to the (patho-)physiological changes during ageing, that is, a blunted chronotropic response. A better understanding of the association between blood pressure and HR helps to assess the usefulness of HR as a vital sign to detect hypotension.

2. What does this study attempt to show?

This study investigated the association between systolic blood pressure (SBP) and HR in multiple cohorts and subgroups in different settings and times.

3. What are the key findings?

No association was found between blood pressure and HR in any age category or subgroups (suspected infection, trauma, ICU-admitted patients). An association between SBP and HR did not exist using multiple measurements per patient of a cohort including ED patients with a suspected infection who were hospitalized.

4. How is patient care impacted?

HR may be an unreliable vital sign to screen for physiological derangement and hypotension in ED patients of all age categories, whereas guidelines such as Advanced Trauma Life Support continue to use HR as one of the vital signs to classify the degree of hemorrhagic shock. Emergency physicians may be misled by traditional concepts about HR disturbances because tachycardia may be absent in hypotension.