

The association between temperature, heart rate, and respiratory rate in children aged under 16 years attending urgent and emergency care settings

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Background and importance Body temperature is considered an independent determinant of respiratory rate and heart rate; however, there is limited scientific evidence regarding the association. This study aimed to assess the association between temperature, and heart rate and respiratory rate in children.

Objective The objective of this study was to validate earlier findings that body temperature causes an increase of approximately 10 bpm rise in heart rate per 1 °C rise in temperature, in children aged under 16 years old.

Design A prospective study using anonymised prospectively collected patient data of 188 635 attendances, retrospectively extracted from electronic patient records.

Settings and participants Four Emergency or Urgent Care Departments in the North West of England. Participants were children and young people aged 0–16 years old who attended one of the four sites over a period of 3 years.

Outcome measures and analysis Multiple linear regression models, adjusted for prespecified confounders (including oxygen saturation, heart rate, respiratory rate, site of attendance, age), were used to examine the influence of various variables on heart rate and respiratory rate.

Main results Among the 235 909 patient visits (median age 5) included, the mean temperature was 37.0 (SD, 0.8).

Mean heart rate and respiratory rate were 115.6 (SD, 29.0) and 26.9 (SD, 8.3), respectively. For every 1 °C increase in temperature, heart rate will on average be 12.3 bpm higher (95% CI, 12.2–12.4), after accounting for oxygen saturation, location of attendance, and age. For every 1 °C increase in temperature, there is on average a 0.3% decrease (95% CI, 0.2–0.4%) in respiratory rate.

Conclusion In this study on children attending urgent and emergency care settings, there was an independent association between temperature and heart rate but not between temperature and respiratory rate. *European Journal of Emergency Medicine* 29: 413–416 Copyright © 2022 The Author(s). Published by Wolters Kluwer Health, Inc.

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Introduction

It is widely accepted that an increase in body temperature will be associated with an increase in heart rate [1,2]. In children, this increase has been suggested to be 10 beats per minute (bpm) per 1 °C rise in temperature [1,3]. There is currently limited scientific evidence regarding the size of this increase, and there is a need for these earlier findings to be validated in a separate group of children, from a different centre.

Between 10 and 20% of attendances to the children's emergency department (ED) involve pyrexia [4–6].

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Pyrexia, or fever, can be defined as a rise in core body temperature above the normal daily variation. Pyrexia is, most usually, secondary to the body's response to infection, both self-limiting infection and significant illness. With this pyrexia is an associated increase in heart rate, likely due to an increase in metabolic rate [7]. There are, however, other physiological reasons for tachycardia in children presenting to the ED, such as pain, anxiety, or agitation. Body temperature has also previously been found to be an independent determinant of respiratory rate [1].

In a clinical setting, observations are taken to allow clinicians to identify the most unwell children and allow the prompt assessment, investigation, and treatment of their illness or injury. A patient with a raised temperature and a tachycardia could be suffering from a significant

infection or sepsis; however, they could also be tachycardic due to the fever itself, the distress of a fever, or pain from a self-limiting condition such as otitis media. Understanding the association between a rise in temperature and heart rate will aid clinicians in their interpretation of a patient's observations, in the context of each individual's attendance to an emergency or urgent care setting. For example, a patient with a significant infection is likely to have a temperature but a raised heart rate as a compensatory mechanism to ensure adequate oxygen delivery to potentially poorly perfused organs. If temperature's impact on heart rate is removed, it may be possible to work out what the true baseline heart rate is and whether there is a serious underlying condition.

This study aimed to validate earlier findings that body temperature is an independent determinant of heart rate and causes an increase of approximately 10 bpm per 1 °C rise in temperature [1]. Previous research looked at data from 21 033 patients from two sites over a 3-year period, with data on children not sent home from the ED removed, to exclude any patient with haemodynamic shock [1].

The goal of this study was to aid clinicians in understanding whether an apparent tachycardia is likely to be an actual tachycardia, and therefore, the authors considered that any association between heart rate and temperature should apply to an undifferentiated cohort. If such an association is evident, then this acts as a cognitive prompt to establish the cause of the raised heart rate – especially because of the link between tachycardia and other serious illness described in national guidance [8].

Our primary aim was to determine the association between temperature and heart rate in children aged under 16 years old. Our secondary aims were to determine the association between temperature and respiratory rate, the association between oxygen saturation and respiratory rate, and the association between oxygen saturation and heart rate.

Methods

Study design and setting

The sample included patients seen consecutively from three ED and one Urgent Care Centre in the North West of England, UK. Eligible cases were children and young people 0–16 years old who attended one of four hospital sites within one National Health Service (NHS) organisation in Greater Manchester, UK. Study design and protocol was prospective over the whole 3 years (01 October 2017–30 September 2020) to avoid potential bias from seasonal variability. Approval was given by both the data controller's research and innovation department and Caldicott Guardian to proceed with this study without seeking formal research ethics committee approval.

Data collection

The data were collected prospectively and then retrospectively extracted from the electronic patient records

for analysis purposes. Following approval by the NHS organisation's Caldicott Guardian and Data Protection Officer, access was granted to anonymised data from the ED and Urgent Centre electronic patient records, to extract the physiological data necessary for this study (as well as the sex, date of birth, and date of attendance, from which the exact age of the child could be calculated).

Data processing

Data were cleaned and anonymised by a data manager in the Research and Innovation Department before passing the data onto the study statistician. As it was the intention to summarise the characteristics from the set of hospitals collectively, as opposed to describing them individually, the data from all four hospitals were combined into one dataset. Values deemed recording errors due to their incompatibility with life were removed.

Data analysis

Descriptive statistics were initially used to examine the distribution of the variables being considered. For each of the research questions, a separate model was used, adjusting for prespecified confounders, though age and location of attendance were included in all. To answer the primary research question, a multiple linear regression model was used with heart rate as the dependent variable. Temperature was included as the independent variable of interest and oxygen saturation, location of attendance, and age were included as prespecified confounders.

For each of the secondary research questions, this methodology was repeated, replacing the variables as appropriate. It is not appropriate to report *P*-values which, given the very large sample size, would be deemed significant regardless of whether the effect size was at all meaningful. Assumptions for linear regression were assessed and for the secondary research questions, respiratory rate was log-transformed to meet the required assumptions. This necessitates that the results for these aims are interpreted in terms of a percentage change in the outcome rather than a raw unit change.

Where the association was of a clinically meaningful magnitude, the strength of interaction between age and the independent variable was also investigated. This investigation was achieved by first fitting the previously described regression model with an added interaction term between age and the independent variable of interest, and then by examining the marginal means at each age.

Analysis was conducted in Stata MP 14 (StataCorp LLC, College Station, Texas., USA)

Results

Data on 235 909 patient visits were recorded. The median age was 5 years (interquartile range, 1–10 years) and the sample was 45% female and 55% male. The four sites

provided differing number of records (35.7, 34.2, 17.0, and 13.1%). Limited demographic characteristics are shown in Table 1. The number of respondents included in each analysis was determined by how many had data recorded for all of the variables in that model.

For the primary analysis, $n = 188\,635$ patients had information on the necessary variables. This and all other results to the research questions are shown in Table 2. The coefficients for the confounder variables are not reported in this table as these are not of interest in answering the research questions.

For every 1 °C increase in temperature, heart rate will on average be 12.3 bpm higher [95% confidence interval (CI), 12.2–12.4], after accounting for oxygen saturation, location of attendance, and age. There is evidence of some limited interaction between age and temperature, meaning the estimate differs depending on the age of the child. The marginal means are reported in Table 3, which gives the amount of change in heart rate with a 1 °C increase in temperature while holding age constant at different values. The marginal means suggest that the influence of temperature on heart rate ranges from a 13.7 bpm increase for a 1 °C temperature increase for the youngest children down to an 8.7 bpm increase for a 1 °C temperature increase for the oldest children.

After accounting for heart rate, location of attendance, and age, there is essentially no influence of temperature on respiratory rate. For every 1 °C increase in temperature, there is on average a 0.3% decrease (95% CI, 0.2–0.4%) in respiratory rate.

Similarly, for every one-percentage-point increase in oxygen saturation, respiratory rate will on average be 1.8% lower (95% CI, 1.8–1.9%), after accounting for heart rate, location of attendance, and age.

For every 1%-point increase in oxygen saturation, heart rate is on average 0.9 bpm lower (95% CI, 0.9–1.0), after accounting for respiratory rate, location of attendance, and age.

As these secondary outcomes did not exhibit clinically meaningful differences, interaction terms were not explored.

Discussion

This study is in keeping with previous research in this area suggesting a rise of 10 bpm increase in heart rate

for every degree rise in temperature. At the time of this study's results being analysed, this is the largest examination (188 635 patient attendances) of the association between temperature and heart rate in a UK nonspecialist setting of a cohort of undifferentiated children attending for urgent or emergency care.

The CI of the results in this study suggests a slightly higher change in heart rate (by 2 bpm) for every 1 °C rise in temperature, than that which was reported in a previous study involving 21 033 patients [1]. The clinical significance of this slightly different finding is questionable; however, it has been possible to investigate the earlier-reported association in a much larger sample.

This study's results from a very large cohort of patients, a decade on from the previous work by Davies and Maconochie [1], and from different sites in a different part of the UK, would suggest that the utilisation of this principle should become more widespread. Current guidance in England and Wales for the identification of serious bacterial illness in children (National Institute for Health and Care Excellence Guidance 51: Sepsis and 143: Feverish illness in children) suggests a direct association between heart rate and risk of infection as part of screening and identification processes. However, studies have demonstrated that this guidance is unspecific and leads to the potential overtreatment with the risks of increased antimicrobial resistance [9,10]. The addition of a correcting variable for fever may improve the specificity of the guidance, and while it is noted that a previous study didn't demonstrate such an association [11] between serious illness and corrected heart rates, the demographics of

Table 1. Data availability from the four sites in this study

Variable	Summary	Min–Max	Number
Age			
Median age in years (IQR)	5 (1–10)	0–15	235 909
Sex			
Male	129 703 (55.0%)	N/A	235 899
Female	106 196 (45.0%)		
Attendances at each site: number (%)			
Site 1: Emergency Department	30 894 (13.1%)	N/A	235 909
Site 2: Emergency Department	84 104 (35.7%)		
Site 3: Urgent Care Centre	40 179 (17.0%)		
Site 4: Emergency Department	80 732 (34.2%)		
Heart rate (beats per minute): mean (SD)	115.6 (29.0)	50–255	191 292
Temperature (°C): median (IQR)	36.8 (36.5–37.3)	25.2–45	192 325
Respiratory rate (breaths per minute): median (IQR)	25 (22–30)	9–90	192 147
Oxygen saturation (%): median (IQR)	99 (98–100)	40–100	191 256

IQR, interquartile range.

Table 2. Coefficients of interest from regression models, stratified by research question

Dependent variable	Independent variable	Coefficient (95% CI)	Adjusted for	Number
Heart rate	Temperature	12.33 (12.24–12.43)	Oxygen saturation, Site, Age	188 635
Respiratory rate (log-transformed)	Temperature	–0.003 (–0.003 to –0.002)	Heart rate, Site, Age	188 670
Respiratory rate (log-transformed)	Oxygen saturation	–0.019 (–0.019 to –0.018)	Heart rate, Site, Age	189 957
Heart rate	Oxygen saturation	–0.91 (–0.96 to –0.86)	Respiratory rate, Site, Age	189 957

CI, confidence interval.

Table 3. Marginal means and temperature coefficient

Age (years)	Temperature coefficient (95% CI)
0	13.71 (13.58–13.85)
1	13.38 (13.26–13.50)
2	13.04 (12.93–13.15)
3	12.71 (12.61–12.80)
4	12.37 (12.27–12.46)
5	12.03 (11.94–12.13)
6	11.70 (11.59–11.80)
7	11.36 (11.25–11.48)
8	11.03 (10.89–11.16)
9	10.69 (10.54–10.84)
10	10.35 (10.19–10.52)
11	10.02 (9.83–10.20)
12	9.68 (9.48–9.89)
13	9.35 (9.12–9.57)
14	9.01 (8.76–9.26)
15	8.67 (8.40–8.94)

CI, confidence interval.

attendances and vaccination schedules have altered significantly in the 10 years since that work was published.

The persistence of a general trend of a 10–12 bpm rise in heart rate for each 1 °C rise in temperature with tight CIs suggests a new lens with which to look at heart rate in relation to illness identification. The fact that the association doesn't hold for respiratory rate may indicate either a difference in the accuracy of the methods of taking the observation (digital probes for heart rate versus manual counting for respiratory rate) or that heart rate is a less dynamic variable than originally thought.

Limitations

This study has several limitations. First, as with all studies of this type, there are certain to be several confounders we do not have data on and so cannot adjust for, potentially biasing the results. Second, these measures, especially heart and respiratory rate, are known for their potential to vary considerably from minute to minute, which introduces an unaccountable element of randomness to the data. Third, respiratory rates were calculated manually, which is known to be less accurate than other methods, though this is internationally standard practice. Fourth, results in this study are only applicable for nonextreme values. Taking temperature as an example, less than 1% of the dataset had a temperature below 35.8 °C. Below this temperature, it is therefore not possible to determine whether the association between temperature and heart rate is linear; therefore, it cannot be suggested what influence a temperature lower than 35.8 °C may have on heart rate. However, such patients are typically identified as being potentially critically unwell from the outset.

Conclusion

After accounting for appropriate confounders, there is a clinically significant association between heart rate and temperature, but no clinically significant influence of temperature on respiratory rate, oxygen saturation on heart rate, or oxygen saturation on respiratory rate.

What is already known on this topic

- Body temperature is an independent determinant of respiratory rate and heart rate.
- Previous studies suggest a rise in heart rate of 10 bpm for every 1 °C increase in temperature.
- There is currently limited scientific evidence regarding the size of this increase.

What this study adds

- For every 1 °C increase in temperature, heart rate will on average be 12.3 bpm higher.
- The influence of temperature on heart rate is age-dependent and ranges from 8.7 to 13.7 bpm increase for a 1 °C temperature increase.
- After accounting for age, heart rate, and emergency care location, there is essentially no influence of temperature on respiratory rate.

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Conflicts of interest

There are no conflicts of interest.

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