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Clinical paper

Heart rate and QRS duration as biomarkers predict the immediate outcome from pulseless electrical activity

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Abstract

Introduction: Pulseless electrical activity (PEA) is commonly observed in in-hospital cardiac arrest (IHCA). Universally available ECG characteristics such as QRS duration (QRSd) and heart rate (HR) may develop differently in patients who obtain ROSC or not. The aim of this study was to assess prospectively how QRSd and HR as biomarkers predict the immediate outcome of patients with PEA.

Method: We investigated 327 episodes of IHCA in 298 patients at two US and one Norwegian hospital. We assessed the ECG in 559 segments of PEA nested within episodes, measuring QRSd and HR during pauses of compressions, and noted the clinical state that immediately followed PEA. We investigated the development of HR, QRSd, and transitions to ROSC or no-ROSC (VF/VT, asystole or death) in a joint longitudinal and competing risks statistical model.

Results: Higher HR, and a rising HR, reflect a higher transition intensity ("hazard") to ROSC ($p < 0.001$), but HR was not associated with the transition intensity to no-ROSC. A lower QRSd and a shrinking QRSd reflect an increased transition intensity to ROSC ($p = 0.023$) and a reduced transition intensity to no-ROSC ($p = 0.002$).

Conclusion: HR and QRSd convey information of the immediate outcome during resuscitation from PEA. These universally available and promising biomarkers may guide the emergency team in tailoring individual treatment.

Keywords: In-hospital cardiac arrest, Prognostics, ECG, Pulseless electrical activity

Introduction

During resuscitation from cardiac arrest, the potential for obtaining return of spontaneous circulation (ROSC) is important and may influence treatment decisions. Such considerations are often based on static factors (i.e., arrest etiology and context, patient comorbidity and presenting rhythm), which by themselves do not reflect treatment response. Dynamic intra-arrest factors such as end-tidal carbon dioxide ($EtCO_2$),[1] cardiac ultrasound[2] and cerebral oxygen saturation[3] have been proposed as prognostic markers, but international guidelines[4] only suggest the use of $EtCO_2$ in this respect.

Heart rate (HR) and QRS duration (QRSd) are biomarkers that are in principle universally available both during the initial and subsequent rhythm checks. The impact of HR and QRSd during the initial rhythm check on survival from pulseless electrical activity (PEA) is unclear. Ho et al, Hauck et al and Bergum et al found no correlation between initial QRSd, HR and survival.[5–7] Weisser found a correlation between initial HR and survival, but not with QRSd.[8] Kim found a correlation between initial QRSd and survival but not with HR,[9] while Aufderheide found a correlation between both HR, QRSd and survival among several studied ECG characteristics.[10]

Skjeflo et al stratified patients with PEA according to whether they achieved ROSC or were declared dead, and investigated the changes in HR and QRSd over the last 12 min preceding these events.[11] Patients who achieved ROSC experienced increasing HR and decreasing QRSd, while those who were declared dead experienced the opposite. It is difficult to apply these results for predictions, however, since they were retrospective and conditional on the outcome.

Joint models have been used to examine the relationship between changes in one or several biomarkers over time and disease outcome.[12] The relationship between the longitudinal

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development of HR and QRSd and the immediate development of ROSC can be studied within this framework.

The aim of this study was to investigate how HR and QRSd as biomarkers are related to the immediate probability of any ROSC during resuscitation from PEA.

Materials and methods

Study setting and population

This was a prospective observational study of in-hospital cardiac arrest (IHCA) among adults, partly due to a quality assurance initiative, that included ECG signals. We reviewed 381 episodes of CA from three hospitals: St. Olav University hospital in Norway (2018–2021), the Hospital of the University of Pennsylvania, USA (2008–2010), and the Penn Presbyterian Medical Center, USA (2008–2010). We also included 74 episodes with primary PEA from St. Olav's hospital registered between 2009 and 2012, annotated and included in a previous study.[11]

Data collection and processing

Defibrillators from different manufacturers (see supplementary material) recorded ECG and impedance during cardiopulmonary resuscitation (CPR) from which chest compressions (including pauses) and ventilations could be determined. All episodes were manually assessed and annotated using a custom-made graphical application in MATLAB.[13]

We evaluated cardiac arrest rhythms and ECG only during chest compression pauses, as compressions introduce noise. PEA was defined as an organized rhythm and different from ROSC if interrupted by chest compressions within one minute, suggesting lack of clinical signs of spontaneous circulation.[14] Temporary ROSC (tROSC) lasted less than 20 min whereas sustained ROSC (sROSC) lasted more than 20 min; the latter defined the end of an episode even if a new arrest ensued. Time of death was defined as the time of the last chest compression or defibrillation attempt if the patient did not obtain ROSC. A detailed description of the data collection process and annotation of arrest rhythms can be found in supplementary material.

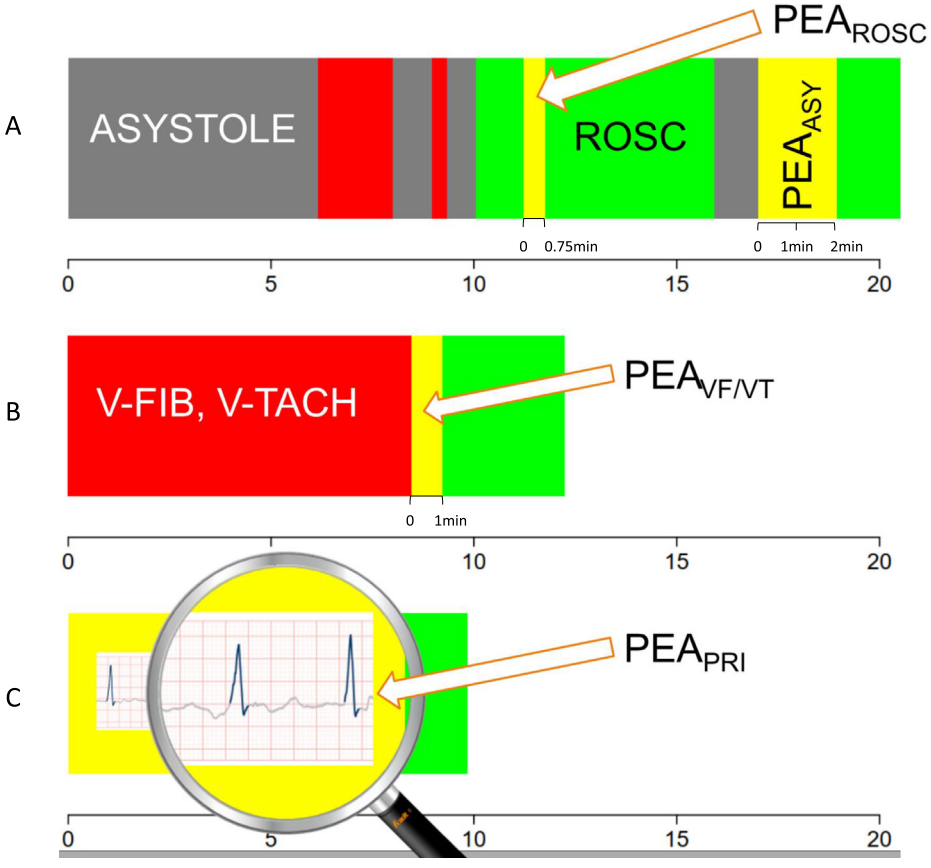


Fig. 1 – Illustration of three episodes (A-C) of cardiac arrest, showing all four types of PEA segments (yellow with arrows). Incidentally, all ended in ROSC. Also illustrated are the two different timelines at work; episode time from start of resuscitation, and PEA segment time in which the clock is "reset" to 0. Color coding: Asystole (gray), VF/VT (red), PEA (yellow), ROSC (green). Episode A contains two PEA segments from different origins. Initially we see transitions between asystole and VF/VT, before ROSC at 10 min. The patient rearrests with PEA_{ROSC} at approximately 12 min. At approx. 16 min the patient enters asystole and gains PEA_{ASY} at approx. 17 min, then regaining ROSC at 19 min. Episode time is cut at 20 min. Episode B shows a transition from VF/VT to $PEA_{VF/VT}$ at approx. 9 min and a transition from $PEA_{VF/VT}$ to ROSC at about 9 min. Episode C illustrates a direct transition from PEA_{PRI} to ROSC at 8 min. As this is primary PEA, segment time coincides with episode time.

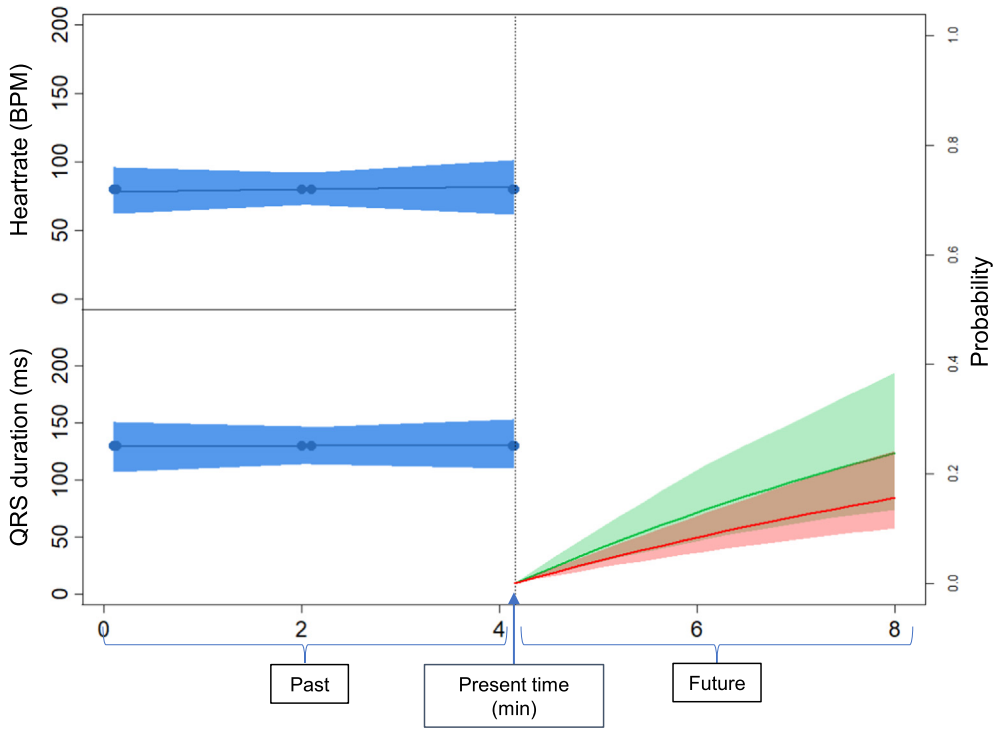


Fig. 2 – Illustration of the "typical patient" who has been in PEAPRI for approx. 4 min. During this period, HR has been constant at 80 bpm and QRSd has been constant at 130 ms (blue dots) as illustrated by the left part of the figure. The right part of the figure illustrates the probabilies of the different immediate outcomes (state following the PEA segment). The probability of ROSC the next 4 minutes is illustrated by the green line with 95% credible interval (green area), the estimated probability of noROSC is illustraded by the red line with 95% credible interal (red area). The estimated probability of remaining in PEA is not shown but equals 1 minus the sum of ROSC and noROSC. The calculation of these probabilities is based on the slope/changes in HR and QRSd up to present time (slope eqals zero in this case), the absolute value of HR and QRSd at present time (80 bpm and 130 ms), the estimated trajectory of HR and QRSd the next 4 minutes (extending the blue line, also at 80 bpm and 130 ms) together with PEA type and time from episode start.

A PEA segment was defined from where PEA was first observed until a transition to another clinical state (ROSC, VF/VT, ASY, or death) was noted. One episode of cardiac arrest may thus contain several PEA segments, and one patient may experience more than one episode. Fig. 1 illustrates transitions in three episodes of cardiac arrest from two patients: showing a total of four PEA segments.

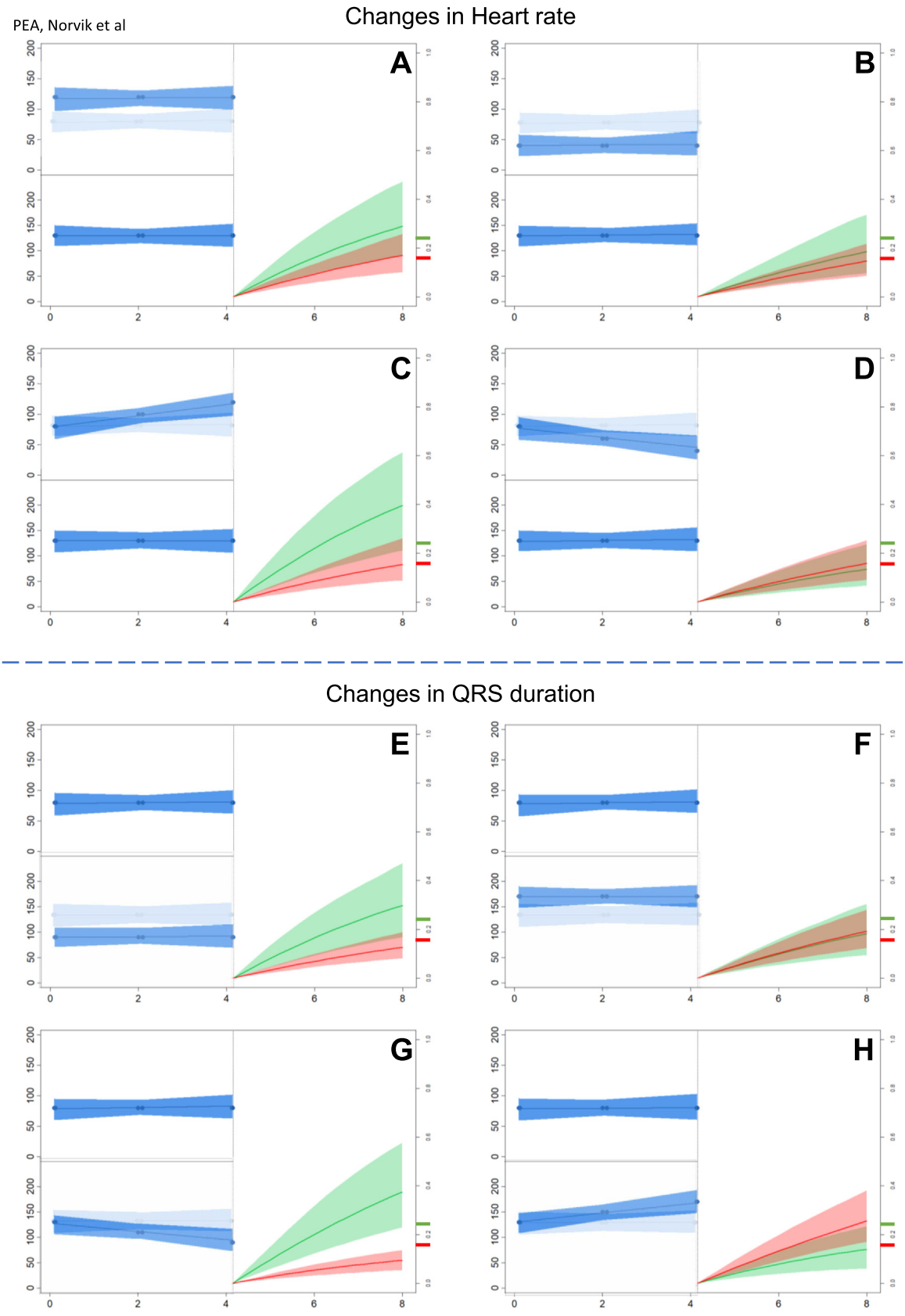
HR and QRSd were determined for all available QRS complexes of every PEA segment by the first author (AN). Start of QRS was defined as a sudden upwards or downwards deflection from a stable baseline. The end of QRS was defined at the J-point, i.e. first part of deflection on the terminal upstroke or downstroke of the QRS.[15] In cases where the J-point could not be defined, the end was defined where the downstroke of the R-wave or the upstroke of the S-wave crossed the baseline. Cases with an unclear J-point were reviewed with an electrophysiologist for adjudication (JPL). HR in beats per minute (bpm) was determined by dividing 60,000 by the RR-time in milliseconds (i.e., time from one R-wave to the next).

Statistical analysis

A joint model relates the longitudinal development of one or more covariates over time to a time-to-event outcome of interest. In the current study, we simultaneously considered the *longitudinal development* of HR and QRSd using linear mixed effect models, and the *time to events* ROSC or no-ROSC (VF/VT, ASY or death) as competing risks; using Cox models in a Bayesian framework. We included both the actual value of HR and QRSd and their estimated slopes. In S1 (supplementary material) we illustrate calculation of the average HR change over time (ΔHR/Δtime, i.e., slope) in one PEA segment. For a thorough introduction to joint models, we refer to supplementary material, Baart et al., Cekic et al. and Elashoff et al.[16–18] We employed the package JMBayes2 version 0.3-0[19] in R version 4.2.1.[20]

A "clock reset" model governed the timeline of the segments, meaning that each PEA segment started at time zero regardless of whether PEA was a primary or secondary rhythm (Fig. 1). Time since start of the episode was added as a fixed covariate along with type of PEA[21]; PEA as the primary rhythm (PEA_{PRI}), PEA following a period of ROSC (PEA_{ROSC}), PEA following VT/VF ($PEA_{VF/VT}$) or PEA following asystole (PEA_{ASY}) (Fig. 1).

The basic outcome measures were the *transition intensities* from PEA to ROSC, and to no-ROSC (equivalent to *hazard rates* in conventional survival models). A transition intensity is the immediate probability that the patient moves to another clinical state in a short time (e.g., the next minute) given the state he or she is currently in.


PEA, Norvik et al
Changes in Heart rate
A
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Changes in QRS duration
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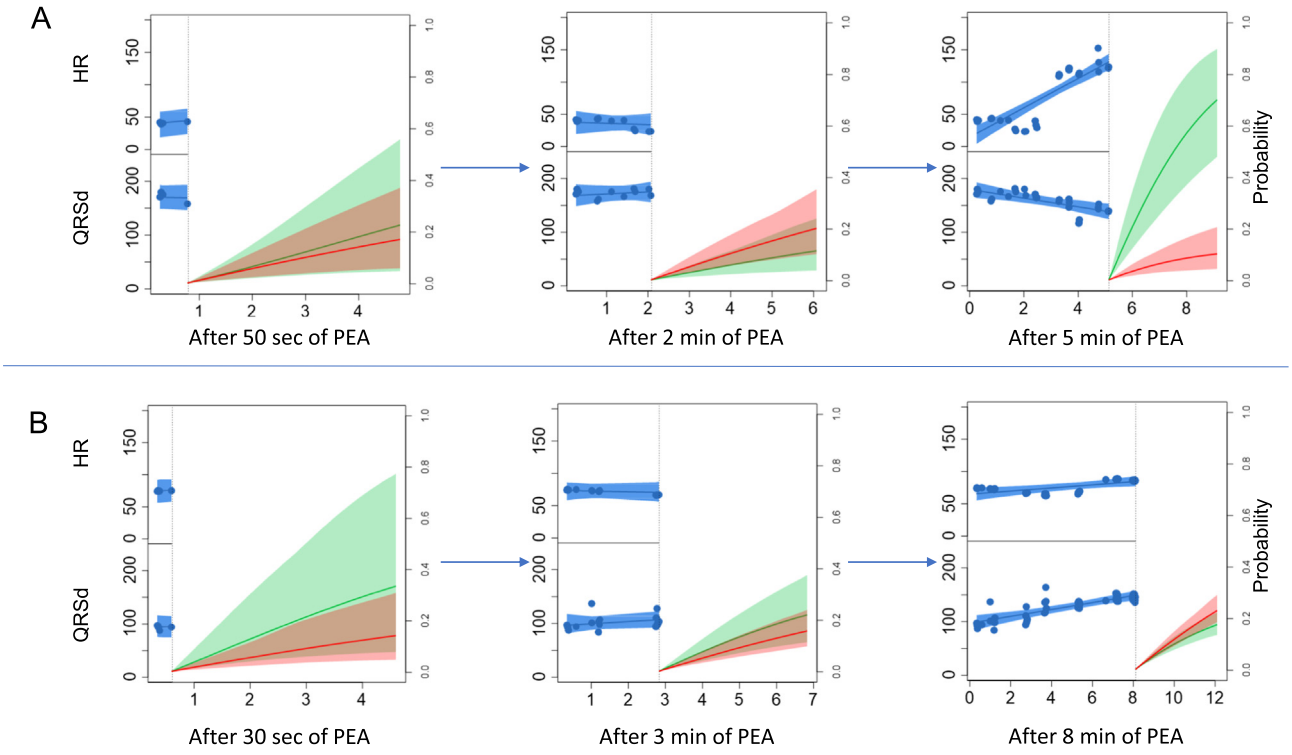

Fig. 4 – PEA segments from two patients that illustrate actual but different HR and QRSd developments in PEA_{PRI} during CPR. Patient A in the upper row gained temporary ROSC at approximately 7 minutes, but rearrested and was later declared dead. Patient B, in the lower row, transitioned to sustained ROSC at 8 minutes and 25 seconds; illustrating that resuscitation should not be terminated based on this observation alone.

Dynamic prediction plots (Figs. 2–4) were used to illustrate the clinical application of the model by showing the estimated probability of ROSC and noROSC the next 4 min (right part). These estimations are deducted from past observations of HR and QRSd (left part) and the estimations are updated as new values are added. To roughly assess the models' ability to predict ROSC within the next 4 min, we calculated the area under the receiver operating characteristic (ROC) curve and positive predictive values using the 559 PEA segments.

Ethics

The Regional Committees for Medical and Health Research Ethics in central Norway approved the study (reference number 2019/785). The need for consent was waived by the committee for patients from the two North American hospitals and for 72 of the patients included from the Norwegian hospital. The remaining patients provided written consent personally or through a next-of-kin. Data were further analyzed anonymously.

Results

A total of 455 episodes from 405 patients comprised the initial dataset. We excluded episodes with noisy or lacking ECG signal, episodes without PEA segments, episodes with an active pacemaker during resuscitation, and episodes containing PEA segments with only one analyzable QRS complex (S3 in supplementary material). From the remaining 327 episodes we extracted 559 segments of PEA in 298 patients (Fig. 1). The median age was 68 years (quartiles 57, 78) and 167 patients were male (56%). Cardiac etiology was presumed in 139 (49%) episodes, 235 (73%) episodes occurred in units with continuous monitoring, and adrenaline was administered in 266 (88%) episodes. Sustained ROSC was achieved in 175 episodes (54%), and 35 patients (12%) eventually survived to discharge (Table 1).

Any ROSC developed in 282 PEA segments, one segment remained in PEA, 70 transitioned to VF/VT, 108 to ASY, and death was declared at the end of 98 segments. The median duration of

Fig. 3 – A. The impact of 40 bpm higher HR. HR and QRSd values from the reference segment can be seen as light shadows. The estimated probability of ROSC and no-ROSC of the reference segment can be seen as thick green and red lines on the probability axes. Fig. 3B. The impact of 40 bpm lower HR. Fig. 3C. The impact of a HR increasing by 10 bpm/min combining the effect of a higher absolute value and change in HR over time. Fig. 3D. The impact of a HR decreasing by 10 bpm/min. Fig. 3E. The impact of a 40 ms lower QRSd. Fig. 3F. The impact of a 40 ms higher QRSd. Fig. 3G. The impact of a QRSd decreasing by 10 ms/min. Fig. 3H. The impact of a QRSd increasing 10 ms/min.

Table 1 – Descriptive data.

Patient characteristics (total 298)	
Male (proportion)	167 (56%)
Median age (quartiles)	68.0 years (quartiles 57–78)
Patients surviving to discharge	35 (12%)
- PEA as primary rhythm	23
- VF/VT as primary rhythm	12
- ASY as primary rhythm	0
Episode characteristics (total 327)	
Cardiac etiology	139 (49%, 41 missing)
Adrenaline administration	266 (88%, 24 missing)
Arrests in units with continuous monitoring	235 (73%, 6 missing)
Median chest compression rate	110 (quartiles 104–115)
Median chest compression fraction	0.88 (quartiles 0.81–0.92)
Presenting rhythm	
- PEA	257 (79%)
- VF/VT	44 (12%)
- ASY	26 (8%)
Episodes ending in sustained ROSC	175 (54%)
PEA segment characteristics (total 559)	
Median duration of PEA segment	225 sec (quartiles 105–415 s)
Median number of QRS complexes per segment	18 (quartiles 7 – 48.5)
Median HR	86.9 (quartiles 54.5 – 120.0)
Median QRSd	135.1 (quartiles 103 – 172)
Median slope (HR (quartiles), QRSd (quartiles))	
- Segments ending in sROSC (n = 145)	3.9 (0.4–8.9), − 0.1 (-3.7–2.5)
- Segments ending in tROSC (n = 137)	2.4 (-0.6 – 6.8), −0.2 (-3.7–2.4)
- Segments ending in VF/VT (n = 70)	3.4 (-0.2 – 7.7), 0.3 (-3.7 – 3.7)
- Segments ending in ASY (n = 108)	1.4 (-2.0 – 5.3), 0.5 (-3.8 – 4.6)
- Segments ending in death (n = 98)	0.8 (-1.5 – 4.2), 0.7 (-1.3 – 3.7)

Table 2 – The association between changes in HR, QRSd and the transition intensity ratios (PEA to ROSC and PEA to noROSC). Note that the effect is linear across the range of HR and QRSd values and slopes. PEA class also affects outcome. In addition, we see that the secondary PEA types get a time penalty as they start later in the episode compared to PEA_{PRI}. E.g, PEA_{ASY} has the same fixed intensity of ROSC as PEA_{PRI}, but since PEA_{ASY} always occurs later in the episode and the transition intensity to ROSC decreases with time – so will also the PEA_{ASY} to ROSC transition intensity. bpm = beats per minute, HR = heart rate.

	PEA to ROSC transition intensity ratio, (95% credibility interval and P-value)	PEA to noROSC transition intensity ratio, (95% credibility interval and P-value)
HR, per 40 bpm (value)	1.39 (1.21–1.58) (<0.001)	1.07 (0.91–1.26) (0.387)
QRS duration, per −40 ms (value)	1.26 (1.13–1.40) (<0.001)	0.84 (0.78–0.91) (<0.001)
HR slope, per 10 bpm min^{-1} (increase)	1.37 (1.15–1.64) (<0.001)	0.84 (0.69–1.14) (0.349)
QRSd slope, per 10 ms min^{-1} (decrease)	1.18 (1.02–1.35) (0.023)	0.76 (0.65–0.89) (0.002)
PEA_{ROSC}	2.48 (1.47–4.13) (0.001)	0.88 (0.49–1.54) (0.688)
$PEA_{VF/VT}$	1.92 (1.25–2.92) (0.003)	2.17 (1.43–3.24) (<0.001)
PEA_{ASY}	1.10 (0.71–1.66) (0.660)	2.04 (1.45–2.87) (<0.001)
Time from episode start (minute, only applicable to $PEA_{VF/VT}$, PEA_{ROSC} and PEA_{asy})	0.98 (0.966–0.998) (0.028)	1.01 (1.004–1.024) (0.008)

the PEA segments was 225 s (quartiles 105, 415 s) (S2). In total 20,344 QRS complexes were included in the analysis. S4 and S5 in the supplementary material visualizes the individual linear developments of HR and QRSd for all PEA segments.

The overall results are displayed in Table 2 and in the dynamic prediction plots in Figs. 2 and 3A-H, as well as in Animations 1 and 2 in supplementary material. To illustrate, we define a "typical patient" as having primary PEA with HR of 80 bpm and QRSd of 130 ms (close to the global average HR and QRSd) that remained constant over time, i.e., with slopes of zero (Fig. 2). A PEA segment with a HR of *40 bpm higher* than 80 bpm (i.e., 120 bpm) has a 1.39 (1.21–1.58) times higher transition intensity to ROSC, and a (non-significant) 1.07 (0.91–1.26) times higher transition intensity to no-ROSC (Table 2). This yields an increased probability of ROSC, an

essentially unchanged probability of no-ROSC (Fig. 3A), and (implicitly) a decreased probability of remaining in PEA. Also, a shrinking QRSd reflects an increased transition intensity to ROSC and a decreased intensity to no-ROSC (Table 2 and Fig. 3E).

The rate of change in HR and QRSd (the slope) also reflect the transition intensity to ROSC. A *10 bpm/*min higher HR slope is related to an increased transition intensity to ROSC by 1.37 (1.15–1.64) times, and a non-significant decrease in the intensity to noROSC (Fig. 3C). A lower QRSd slope is related to an increased transition intensity to ROSC and a decreased intensity to no-ROSC (Fig. 3G).

Type of PEA is also related to the transition intensities. With PEA_{PRI} as reference, PEA_{ROSC} and $PEA_{VF/VT}$ reflect an increased transition intensity to ROSC. PEA_{ASY} is not by itself associated with changes in the transition intensity to ROSC compared to PEA_{PRI}. But due to the accumulated time since start of the episode, PEA_{ASY} has a worse outcome as every additional minute is associated with decreased transition intensity to ROSC and increased transition intensity to no-ROSC.

A high QRSd and a positive QRSd slope reflect an increased probability of no-ROSC (Fig. 3F and H). Still, 87 segments with a QRSd increase of more than 1 ms/min and 23 segments with a QRSd increase of more than 5 ms/min, gained ROSC (S4 in supplementary material).

Fig. 4 illustrates the clinical application of our model using observation at three timepoints from two actual PEA segments. This is also illustrated by the two videos in the online supplementary material. Animation 1 corresponds to the segment illustrated in Fig. 4A and Animation 2 corresponds to the segment illustrated in Fig. 4B.

The AUC of the ROC curve was 0.74 (CI 0.70–0.78) (S6) when comparing the outcome of each segment with predicted ROSC four minutes ahead. A threshold probability of ROSC higher than 50% yielded a PPV of 80% (Table 1 in supplementary material).

Discussion

To our knowledge, this is the first study to quantify how heart rate and QRS duration may predict the immediate outcome *during ongoing resuscitation* from pulseless electrical activity. Our study stands out by including ECG data throughout the entire episode, by using a proper statistical method that is not conditional on the outcome, and finally by linking both values and changes in the biomarkers as dynamic predictors – as opposed to simple averages – to the outcome.

Clinical implications

An immediately available biomarker allows one to monitor progress of the patient and may allow for personalization of treatment. *How* is a matter for debate. However, while HR is easily observed on the monitor, judging QRS duration may require more sophistication. Our study shows that patients with higher/increasing HR and narrower/decreasing QRSd have higher probability of gaining ROSC. A positive development, as observed in Fig. 4A, could encourage one to carry on resuscitation and not change much, in particular avoiding hands off time. Further research may clarify whether, for example, titrating the dose of adrenaline may also be guided by this biomarker response.

A neutral development as observed in Fig. 4B, may prompt one to reconsider some of the aspects of resuscitation, like evaluating the quality of chest compressions and searching more closely for reversible factors. Further research may clarify if alternative sources of information like focused echocardiography, rSO2 or EtCO2 proves useful here.

A high/increasing QRSd is associated with increased transition intensity to VF/VT, ASY or death. This is a statistically significant result (Table 2), but such development should not be interpreted too strongly and was commonly seen in segments ending in ROSC (S4 in supplementary material). For this reason, QRS widening cannot be used alone to terminate the resuscitation effort, nor be used as the only indication of worsening of the patient's condition (Fig. 4B). However, it is nevertheless a signal that may prompt for re-evaluating the situation. Thus, while significant changes in HR and QRSd following an intervention or altered strategy may provide immediate feedback, the response characteristics (time and degree) by these biomarkers remain to be investigated.

PEA type is also related to the probability of gaining ROSC, as described in our previous paper.[21] It is interesting to note that $PEA_{VF/VT}$ develops quickly into either ROSC or no-ROSC states (VF/VT, ASY and death). This is not a contradiction but expresses the inherent instability of $PEA_{VF/VT}$ compared to primary PEA.

Our results are highly significant. Still, we stress that understanding and quantifying the information obtained by the biomarkers are more important than making precise predictions. While we do not believe that survival can be increased by forcing the HR to increase or the QRS complex to shrink, we find it reasonable that these biomarkers reflect the underlying process (see below). To further improve the utility of HR and QRS measurements beyond inspecting the ECG, one may consider automatic and possibly filtered capture and display of these characteristics on the monitor. Other promising biomarkers like ETCO2, and rSO2 could also be investigated with focus on changes during the episode, rather than merely their average values. The joint model is a powerful tool for this purpose.

The QRS complex reflects changes in the physiological state of the heart

Heart rate and the QRS complex duration respond quickly to changes in the cardiac homeostasis. Several studies have shown that acute myocardial ischemia increases QRS duration and relieving the ischemia normalizes QRS duration immediately. Injecting ceramic microspheres into the left circumflex artery of anesthetized dogs has shown to increase QRSd[22] and Holland et al. reported that the intrinsicoid deflection (time from beginning of QRS complex to the top of the R wave) would increase 2–3 times relative to baseline values after ligating the left descending coronary artery in porcine hearts and return to normal within 2–4 heartbeats after releasing the occlusion.[23]

Investigators have also found exercise-induced QRSd prolongation in humans with angina[24] and significant narrowing of the QRS complex immediately after successful reperfusion in patients with ST-elevation myocardial infarction.[25] Telemetry recordings have shown an increasing QRSd of at least 20 ms prior to arrest in 15 out of 81 IHCA.[26] Continuous QRS widening prior to asystole or PEA due to right ventricular strain has also been described.[27] In addition, hyperkalemia is also known to widen the QRS complex.[28] Changes in QRSd during cardiac arrest has also been described.[11]

The narrowing of the QRS complex observed in our study may be a response to improved myocardial perfusion or relieved right ventricular strain during resuscitation.

HR also changes in conjunction with cardiac arrest. Several investigators have demonstrated bradycardia in patients prior to arrest[26,29] and HR changes during resuscitation possibly due to chest compressions and/or epinephrine.[11,30]

Limitations

Defining ROSC as an organized rhythm without chest compressions for more than one minute is one of the major limitations in this study. Differing between ROSC and PEA in retrospect is difficult without objective measures of circulation. While impedance signals might assist in differentiating ROSC form PEA, this information is not available to the treating team.[31] This definition may have caused an overestimation of temporary ROSC.

Using 1-lead ECG to analyze QRSd is a limitation.[32] A multi-lead ECG would make it easier to capture the true QRS duration. Many of the included QRS complexes have a very pathological appearance with no clear J-point making it difficult to define the end. By consistently measuring the same parts of the QRS complex within each episode one may minimize this measurement bias. Annotations were performed by the first author only, except in cases with morphologically challenging QRS complexes. Confidence in the data would have improved with duplicate revision of the data. We also note that the first author was not blinded to patient outcome during the annotation process.

Only episodes containing PEA segments with at least two measurable electrical heart cycles were included in our study. Also, a linear slope is admittedly a simple model for biomarker development. However, actual values are included, and more complex models were both hard to make converge and to interpret and did not improve diagnostic performance.

The calculation of AUC and PPV was done using the dataset included in the model. This produces optimistic estimates of performance.[33]

Finally, the decision to declare a patient dead is based on several factors and not only the isolated ability of the heart to regain ROSC. Our study may thus be subject to some degree of prognostication bias, since some of the patients declared dead could have achieved ROSC (but not necessarily long-term survival) with longer resuscitation time.[34] This may lead to an underestimation of the impact of HR and QRSd on ROSC achievement. Even though the condition of the patient's heart is improving with increasing HR and decreasing QRSd, the resuscitation effort may still be terminated due to conditions unrelated to the heart.

Conclusion

Heart rate and QRS complex duration conveys information of the immediate outcome of PEA. These are universally available and promising biomarkers that may guide the emergency team in tailoring individual treatment. A high and/or increasing HR and a low and/or decreasing QRS duration suggests imminent ROSC and encourages further efforts. Absence of such changes are nonspecific, however, and should encourage re-evaluation rather than termination of resuscitation.

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Conflicts of interest

All authors state no conflicts of interest.

CRediT authorship contribution statement

A. Norvik: Conceptualization, Methodology, Validation, Formal analysis, Investigation, Data curation, Writing – original draft, Visualization. **J.T. Kvaløy:** Conceptualization, Formal analysis, Methodology, Writing – review & editing, Visualization. **GW. Skjeflo:** Conceptualization, Data curation, Writing – review & editing. **D. Bergum:** Conceptualization, Investigation, Data curation, Writing – review & editing, Supervision. **T. Nordseth:** Conceptualization, Writing – review & editing. **J.P. Loennechen:** Conceptualization, Data curation, Writing – review & editing, Supervision. **E. Unneland:** Data curation, Writing – review & editing. **D.G. Buckler:** Data curation, Writing – review & editing. **A. Bhardwaj:** Writing – review & editing. **T. Eftestøl:** Writing – review & editing. **E. Aramendi:** Software, Writing – review & editing. **BS. Abella:** Writing – review & editing. **E. Skogvoll:** Conceptualization, Methodology, Validation, Formal analysis, Investigation, Data curation, Writing – review & editing, Supervision, Project administration, Funding acquisition.

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Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.resuscitation.2023.109739.

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