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ECG patterns in early pulseless electrical activity-Associations with aetiology and survival of in-hospital cardiac arrest

Daniel Bergum[a,b,d,*], Gunnar Waage Skjeflo[a], Trond Nordseth[b], Ole Christian Mjølstad[a,c], Bjørn Olav Haugen[a,c], Eirik Skogvoll[a,b], Jan Pål Loennechen[a,c]

[a] Department of Circulation and Medical Imaging, Norwegian University of Science and Technology, Trondheim, Norway
[b] Department of Anaesthesia and Intensive Care Medicine, St. Olav University Hospital, Trondheim, Norway
[c] Department of Cardiology, St. Olav University Hospital, Trondheim, Norway
[d] Norwegian Air Ambulance Foundation, Norway

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ABSTRACT

Introduction: Pulseless electrical activity (PEA) is an increasingly common presentation in cardiac arrest. The aim of this study was to investigate possible associations between early ECG patterns in PEA and the underlying causes and survival of in-hospital cardiac arrest (IHCA).

Methods: Prospectively observed episodes of IHCA presenting as PEA between January 2009 and august 2013, with a reliable cause of arrest and corresponding defibrillator ECG recordings, were analysed. QRS width, QT interval, Bazett's corrected QT interval, presence of P waves and heart rate (HR) was determined. QRS width and HR were considered to be normal below 120 ms and within 60–100 cardiac cycles per minute, respectively.

Results: Fifty-one episodes fulfilled the inclusion criteria. The defibrillator was attached after a median of one minute (75th percentile; 3 min) after the onset of arrest. Ninety percent (46/51) had widened QRS complexes, 63% (32/51) were defined as 'wide-slow' due to QRS-widened bradycardia, and only 6% (3/51) episodes were categorized as normal. No unique cause-specific ECG pattern could be identified.

Further 7 episodes with a corresponding defibrillator file, but without a reliable cause, were included in analysis of survival. Abnormal ECG patterns were seen in all survivors. None of the patients with 'normal' PEA survived.

Conclusion: Abnormal ECG patterns were frequent at the early stage of in-hospital PEA. No unique patterns were associated with the underlying causes or survival.

Introduction

The prevalence of pulseless electrical activity (PEA) as the presenting rhythm in cardiac arrest (CA) varies between 17% and 50%, and has in some studies been reported to be increasing.[1–5] PEA occurs both with cardiac and non-cardiac causes.[6–8]

Ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT) are associated with cardiac causes and demand early defibrillation.[6,7] Asystole, defined as no detectable cardiac electrical activity, is probably not suitable to further analysis. However, during PEA, heart rate (HR) and QRS complex abnormalities may provide additional information regarding the probability of achieving return of spontaneous circulation (ROSC) and further survival. Briefly, PEA with a normal electrocardiogram (ECG) is expected to have a prognostic advantage compared to a pathological ECG.[9,10]

Associations between the width of the QRS complex and the underlying mechanism of CA has been suggested.[11] Whether cause-specific clues in terms of heart rate (HR), QRS width and QT interval abnormalities may be found in the initial ECG has not been previously investigated.

The most common 'reversible' causes, often found in non-shockable episodes of CA, are often memorized as '4H4T'; hypoxia, hypovolaemia, hypo-/hyperkalaemia, hypothermia, thromboembolism (pulmonary embolus), toxins, tension pneumothorax, tamponade (cardiac).[12] Identifying these causes may lead to individualized and better treatment.[11,13] In a recent publication, we demonstrated a survival benefit in in-hospital cardiac arrest (IHCA) if the causes were recognized by the in-hospital emergency team (ET). Patient records and pre arrest clinical signs were the

* Corresponding author at: Department of Anaesthesia and Intensive Care Medicine, St. Olav University Hospital, P.O. Box 3250 Sluppen, 7006 Trondheim, Norway.
E-mail address: daniel.bergum@ntnu.no (D. Bergum).

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most frequently utilized information sources when causes were recognized.[14]

A defibrillator with an ECG monitor is almost always available within few minutes after IHCA. Knowledge of specific associations between ECG patterns and causes of CA may be valuable in clinical decision-making during and after resuscitation. This may apply both in the hospitalized setting and out-of-hospital. The aim of this study was to investigate possible associations between early defibrillator ECG patterns in PEA and the underlying causes of IHCA and survival.

Method

Material

All IHCA episodes in adult patients 18 years or older receiving cardiopulmonary resuscitation (CPR) at the St. Olav university Hospital in Norway between January 2009 and august 2013 were prospectively observed. If PEA was the first documented rhythm, a corresponding defibrillator file was identified, and a reliable cause of CA could be identified, the episode was included for further analysis. Details about inclusion strategy, ET organization and a thorough retrospective aetiology investigation have been described in recent papers.[6,14,15] In short, to define a cause as 'reliable', it had to be confirmed, or the alternative causes had to be excluded by objective diagnostic measures pre- or post cardiac arrest.

Defibrillator files were collected from semi-automatic and manual defibrillators at general wards, intermediate critical care units, the emergency department, the intensive care unit, and/or and from the ET trolley: Lifepak®1000 and Lifepak®20 (Physio-Control, Redmond, USA) and Zoll M-Series® (Zoll Medical Corporation, Massachusetts, USA). Whereas Lifepak®1000 is a semi-automatic defibrillator suitable for public access localizations, the Lifepak®20 and Zoll M-Series® are also constructed for professional operators with the option of manual mode and external pacing. We downloaded defibrillator files from the defibrillators through infrared communication ports (Lifepak®1000 and Lifepak®20) or via a 'Linear Flash' card reader (Zoll M-Series®).

PEA characteristics

PEA was defined as any electrical cardiac activity expected to generate pulsatile circulation, where CPR was initiated due to lack of a palpable pulse and general clinical signs of circulatory arrest.

ECG defibrillator files were analysed using software from the manufacturers; Code-Stat™ 9.0 (Physio-Control, Redmond, USA for files generated by the Lifepak®20 and Lifepak®1000) and RescueNet Code Review™ 5.51 (Zoll Medical Corporation, Massachusetts, USA for files generated by the Zoll M-Series®).

Three consecutive QRS complexes, within a 4–8 s long ECG strip from the first pause in chest compressions due to ventilation measures or pulse-check, were selected for further analysis. Between the onset of CA and the first ECG strip analysed, only basic life support was applied, except for ongoing intravenously fluid therapy in some patients. Two electrophysiologists (cardiologists OCM and JPL) independently measured QRS widths and QT intervals. Start and end of the widths/intervals were marked and measured on paper print-outs at 50 mm/s (Physio-Control) and 25 mm/s (Zoll). The ECG paper printouts were photo-scanned and archived. Start of QRS was defined at the first visible deflection of the QRS complex from the baseline. End of QRS was defined as the last visible signals assessed to be a part of the ventricular depolarisation. The end of the T wave was defined at the intersection of the steepest tangent of the descending part of the T wave and the baseline. We used Bazett's formula for RR-correction of the QT intervals: $QTc = QT/\sqrt{RR}$.[16,17] QRS widths and QT intervals from each episode of PEA were averaged from the three consecutive ECG cycles independently measured by each of the two electrophysiologists. The average HR from each PEA episode were determined by the first author based on the available RR-intervals within the ECG-strip analysed.

We considered the QRS complex as normal or 'narrow' if the QRS width was less than 120 milliseconds (ms), and 'wide' otherwise. The HR was considered as 'slow' if below 60/min, normal if within 60–100/min and 'fast' if 100/min or above, based on previous relevant studies.[18] Thus, six categories of PEA patterns were defined: normal, wide, narrow-slow, narrow-fast, wide-slow, and wide-fast.

A P wave was considered as present if it was clearly associated with the QRS complex. If dissociated, and with uncertain presence, then the P wave was not considered present.

The 'Bazetts' QT correction was calculated for all episodes although normal HR-QTc correlations were not expected in episodes with widened QRS due to pathologic repolarisation and thus altered QT intervals.[19]

Statistical analysis

Statistical analyses were performed with STATA/IC 13.1 for Windows (StataCorp. LP, Texas USA). Inter-rater agreements between the two electrophysiologists' QRS- and QT assessments were calculated using kappa statistics for two unique raters. HR, QRS widths and QT intervals are presented as median with inter-quartile range (IQR), by causes and by the survival categories 'no ROSC', '1-h survival' and 'hospital discharge'. Scatterplots of HR against QRS width were then constructed according to cause, and according to one-hour survival.

Ethical aspects

In all episodes considered for inclusion, we requested the patient or a family member next of kin for a written informed consent. The study was approved by the regional committee for medical and health research ethics in central Norway, REK 4.2008.2402, ref. no: 2009/1275.

Results

In 144 of 302 IHCA episodes (48%) PEA was the first presenting rhythm. In 58 of these 144 a defibrillator file was identified available for analysis of PEA patterns and immediate survival beyond one hour, and hospital discharge. In 51 of the 58 episodes one or more reliable causes were identified (Fig. 1). ECGs from these 51 episodes were included in the analyses of PEA patterns and the underlying causes of arrest. In 17 episodes, two causes were identified as the triggering causes of arrest, thus causes were not mutually exclusive.

Median delay from onset of CA to the attachment of a defibrillator was one minute, with the 75th percentile at three minutes. CA was witnessed in 46/51 (90%) and monitored in 23/51 (45%) episodes (Table 2). An extended comparison between characteristics of the 51 included episodes and episodes not included due to missing defibrillator files or no reliable causes identified is shown in Table 2.

The inter-rater agreements between the two electrophysiologists' QRS- and QT-determinations were both 95% with kappa 0.77 and 0.85, respectively ($p < 0.001$ for both).

A P-QRS association was identified by both raters in 18 of 51 episodes (35%). The presence of P-QRS associations, the median of HR, QRS widths, QT- and corrected QT intervals and their respective interquartile ranges are presented for each of the survival categories and for groups of causes in Table 1.

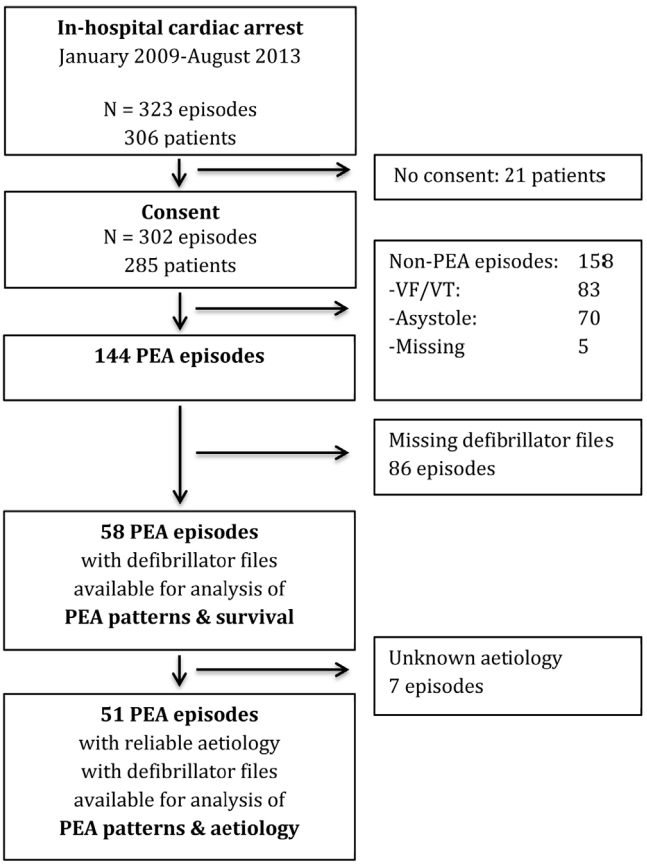

Fig. 1. Data generation flow chart. PEA, pulseless electrical activity; VF/VT, pulseless ventricular fibrillation/tachycardia.

Forty-six/fifty-one episodes (90%) had widened QRS complexes and 32/51 (63%) were defined as wide-slow due to additionally low HR below 60/min. Only 3/51 episodes (6%) were considered as normal. At hospital admission 19/51 (37%) had a normal ECG or a regular peripheral pulse between 59 and 100/min (no ECG recorded at admission). Signs of ischaemia (ST-deviation or bundle branch block) were found in 11/51 (22%), atrial fibrillation in 5/51 (10%), sinus tachycardia in 5/51 (10%), ischemic sequelae (increased Q or T-wave inversion) in 4/51 (8%) and irregular peripheral pulse (no ECG) in 2/51 (4%).

Figs. 2 and 3 show the distribution of causes among the ECG patterns defined. Causes are not mutually exclusive, thus exceeding the number of episodes. Episodes of cardiac, non-cardiac and 'mechanical' origin were found in all ECG categories, except in 'narrow-fast' in which no episodes were observed. The causes of arrest in the three episodes of 'normal' PEA pattern were myocardial infarction with rupture of the ventricle wall, sepsis and aortic dissection of the ascending and the descending aorta.

Following 21/51 episodes (41%), the patients achieved stable ROSC beyond one hour. Following 6/51 episodes (12%), the patients survived to hospital discharge. The HR among the 21 one-hour survivors ranged from brady- to tachycardia (median 48/min, range 25–128/min). Their QRS complexes were wide (median 169 ms, range 79–264 ms), only one episode had a QRS complex narrower than 120ms. Six of the 21 one-hour survivors (29%) had a present P-QRS association. The causes of arrest among the 21 one-hour survivors were nine with hypoxic/pulmonary cause (43%), five with myocardial infarction (24%), three with pulmonary embolus (14%), two with heart failure (10%), two with aortic dissection (10%), one with cardiac tamponade (5%) and one with septic shock (5%). Among the six patients who survived to hospital discharge, there were four with hypoxic/pulmonary causes, one with pulmonary embolus and one with myocardial infarction. Fig. 4 shows 1-h survival according to the ECG patterns defined. Seven additional PEA episodes with a corresponding defibrillator file, but without a reliable cause of arrest, are included in the figure.

Discussion

The main finding in this study is that none of the early ECG patterns considered were uniquely associated with the underlying causes or the underlying mechanism behind the arrest. This is the first study investigating the association between the first presenting ECG characteristics and the causes of CA during PEA. In Table 2, we found that the 51 PEA episodes included in the analysis are roughly comparable to the PEA episodes not included because

Table 1
ECG characteristics in in-hospital cardiac arrest by survival and by causes.

	Number (no, percent)	P-wave (no, percent)	HR (median, IQR)	QRS (median, IQR)	QT (median, IQR)	QTc – Bazett (median, IQR)
Survival (percent of 58 PEA episodes with defibrillator files)						
No ROSC	35 (60%)	13 (37%)	51 (39–63)	167 (125–209)	493 (409–569)	439 (376–508)
1-h survival	17 (29%)	4 (24%)	45 (41–54)	182 (150–235)	540 (465–602)	475 (426–509)
Hospital discharge	6 (10%)	2 (33%)	42 (34–94)	145 (140–174)	528 (377–670)	513 (448–550)
Presence of causes (percent of 51 episodes with defibrillator files and reliable aetiology)						
Cardiac, all ca	26 (51%)	9 (35%)	57 (41–66)	168 (134–239)	518 (404–602)	504 (410–562)
Uses						
Myocardial inf.	13 (25%)	6 (46%)	59 (52–78)	170 (140–242)	520 (340–595)	509 (428–564)
Cardiac tamponade[a]	8 (16%)	1 (13%)	58 (31–73)	166 (125–224)	499 (428–521)	446 (357–512)
Heart failure	5 (10%)	3 (60%)	41 (41–66)	240 (174–252)	595 (400–610)	504 (492–521)
4H & 4T, all causes	33 (65%)	11 (33%)	48 (35–65)	169 (134–199)	492 (412–555)	475 (395–512)
Hypoxia	14 (27%)	5 (36%)	46 (35–60)	170 (142–182)	479 (380–574)	484 (347–515)
Hypovolaemia	6 (12%)	3 (50%)	55 (44–68)	165 (122–227)	494 (430–584)	494 (454–567)
	5 (10%)	2 (40%)	45 (45–54)	169 (134–170)	492 (484–517)	426 (419–448)
Thrombus/pulm.emb.						
Cardiac tamponade[a]						
Other	10 (20%)	3 (30%)	43 (34–60)	165 (125–227)	539 (452–584)	437 (354–488)

ECG, electrocardiography, HR, heart rate, ROSC, return of spontaneous circulation, 4H & 4T, hypoxia, hypovolaemia, hypo-/hyperkalaemia, hypothermia, thrombosis/pulmonary embolus (pulm.emb.), tamponade cardiac, tension pneumothorax, toxins/intoxication, 'Other causes', aortic dissection/rupture, sepsis, gastrointestinal bleeding.

[a] Cardiac tamponade ($n = 8$) is one of the 'reversible' 4H & 4T, but is here presented within cardiac causes due to the main reason of tamponade being ruptured ventricular wall following myocardial infarction. *Causes are not mutually exclusive.*

Table 2
Comparison of baseline characteristics of 144 PEA episodes according to the presence/absence of reliable causes and defibrillator files.

	Reliable causes and defibrillator files ($n = 51$)	Missing defibrillator files or no reliable cause ($n = 93$)
Male (no., percent)	29 (57%)	61 (66%)
Age (median years, IQR)	75 (65–82)	75 (64–82)
CCI, incl age (mean ± SD)	5.4 (2.1)	5.1 (2.2)
CPR delay (median min., 90th percentile)	1 (1)	1 (1)
Defibrillator attached (median min., 90th percentile)	1 (5)	1 (5)
CPR duration (median min., IQR)	20 (15–35)	18 (11–29)
Witnessed (no., percent)	46 (90%)	79 (85%)
Monitored (no., percent)	23 (45%)	43 (46%)
Cum. epinephrine (median mg, IQR)	2 (0–5)	2 (1–4)
Intubation (no., percent)	37 (73%)	57 (61%)
Time to intubation (median min., IQR)	10 (5–15)	10 (8–13)
Time to first ROSC (median min., IQR)	7 (5–14)	11 (6–18)
Time to CPR stopped (median min., IQR)	20 (15–35)	18 (11–29)
CPR stopped because of ROSC (no., percent)	20 (39%)	43 (46%)
Survival >1 h (no., percent)	21 (41%)	45 (48%)
Survival hospital discharge (no., percent)	6 (12%)	13 (14%)
Presence of cardiac causes (no., percent)	26 (51%)	33 (35%)
Presence of '4H & 4T' causes (no., percent)	33 (65%)	45 (48%)
Presence of 'other causes' (no., percent)	10 (20%)	17 (18%)
Recognition of causes by the ET	41 (80%)	53 (57%)

CCI, age-adjusted charlson comorbidity index; CPR, cardiopulmonary resuscitation; Cum. epinephrine, cumulative epinephrine administered; 4H & 4T, hypoxia, hypovolaemia, hypo-/hyperkalaemia, hypothermia, thrombosis/pulmonary embolus, tamponade cardiac, tension pneumothorax, toxins/intoxication;'other causes', aortic dissection/rupture, sepsis, gastrointestinal bleeding; ET, emergency team; no., count; IQR, inter-quartile range; SD, standard deviation.

of unknown aetiology or missing defibrillator files. The most striking differences may be the larger amount of overlapping cardiac and '4H4T' causes among the 51 included episodes, possibly representing more co-morbidity in this group.

There was a substantial amount of pathologic ECG findings with widened QRS complexes in 90% and additional bradycardia in 63%. None of the 1-h survivors had normal ECG patterns and the majority had both widened QRS complexes and bradycardia. Thus, our study does not support the idea of prognostication based on an analysis of the early ECG patterns. However, since only 37% of patients had a normal ECG at hospital admission, inclusion may have been biased so that the 51 episodes in this study are dominated by patient cases with significant cardiac comorbidity.

The overall survival to discharge of 13% from 144 PEA episodes (140 patients) in the total cohort is comparable to other in-hospital studies.[20,21]

We found prolonged median QT intervals with all causes as well as survival groups. This might reflect the beginning of myocardial ischaemia and pathologic repolarisation, as expected in cases where the 'exposure' to CA continuous. This process is perhaps independent of the triggering cause itself. It remains to be seen whether the ECG patterns respond differently to CPR over time.

In a review article from 2014, Littmann et al. suggested – on a theoretical basis – an association between narrow QRS complexes and the most frequent mechanical causes of PEA, and that widened

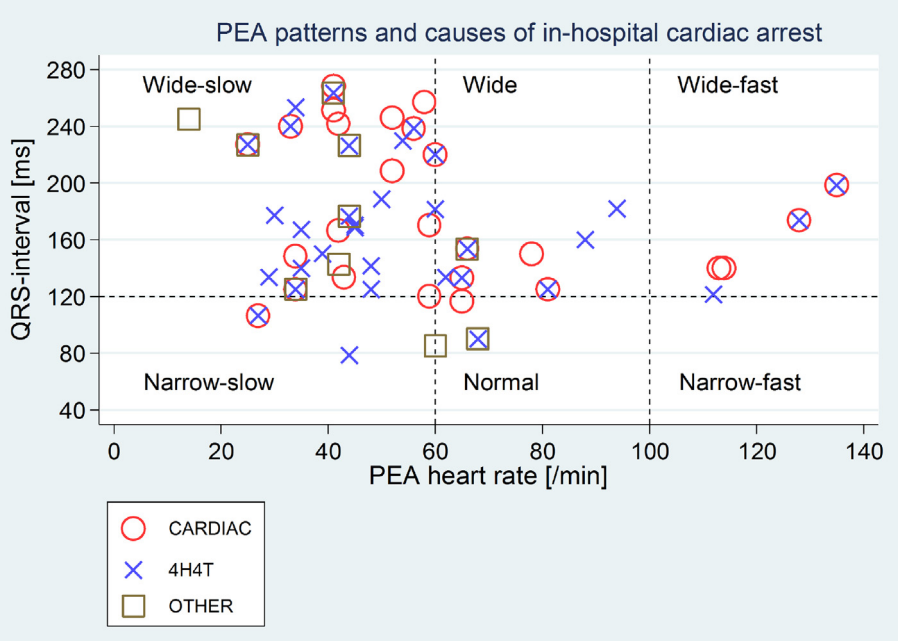

Fig. 2. Reliable causes of pulseless electrical activity (PEA) and patterns of early electrocardiogram (ECG) during resuscitation from in-hospital cardiac arrest. 4H4T, hypoxia, hypovolaemia, hypo-/hyperkalaemia, hypothermia, thromboembolism (pulmonary embolus), toxins, tension pneumothorax, tamponade (cardiac). Other, aortic rupture/-dissection, sepsis, exacerbation of cancer.

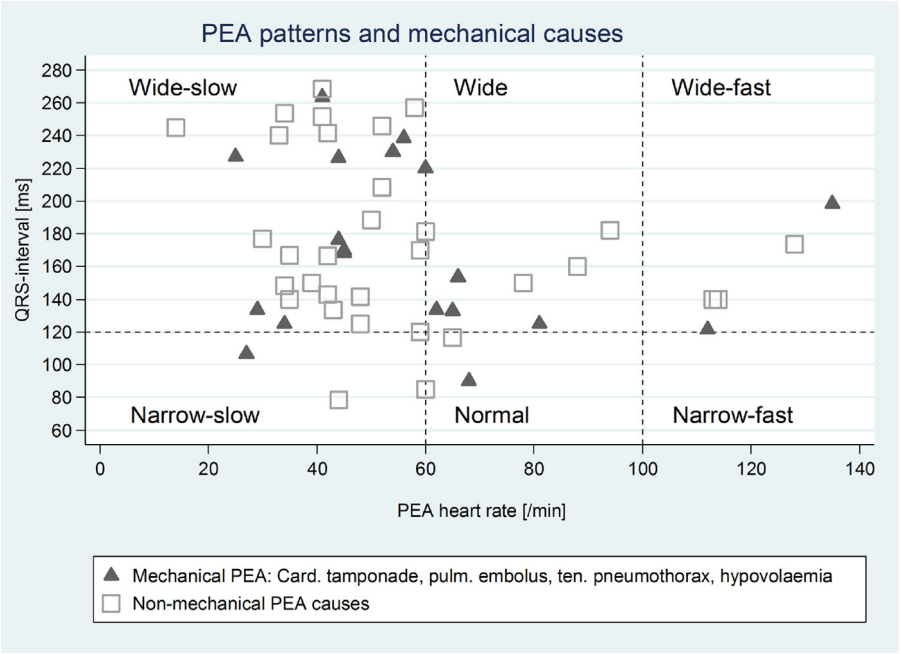

Fig. 3. Mechanical and non-mechanical causes of pulseless electrical activity (PEA) and patterns of early electrocardiogram (ECG) during resuscitation from in-hospital cardiac arrest. Tamp., tamponade; emb., embolus; pn., pneumothorax. Hypovolaemia was considered to be a mechanical cause in cases of sudden loss of blood and a subsequent 'empty heart'.

QRS complexes were associated with metabolic conditions.[11] We could not find support for such an association ('non-mechanical PEA causes' in Fig. 3). The explanation might be that although mechanical causes of CA may not directly affect the myocardial depolarisation at an early stage, a metabolic deterioration induces depolarisation abnormalities as CA progress, especially if there is little response to immediate CPR efforts. An association between ECG pattern and the mechanism of arrest may still exist and may possibly be identified more shortly after the occurrence of the arrest than in this study.

Aufderheide et al. conducted a thorough PEA analysis of defibrillator-based ECG strips in out-of-hospital cardiac arrest (OHCA) episodes.[18] They followed up these analyses in the patients arriving at the hospital alive. Briefly, they found more abnormal ECGs among non-survivors than survivors. They further observed a normalization of arrhythmias, both tachycardia and bradycardia, and an increased presence of P waves during hospitalization among patients who ultimately survived. However, they noted substantially narrower QRS complexes than in the present study, possibly reflecting less myocardial compromise from the 'exposure to CA', or

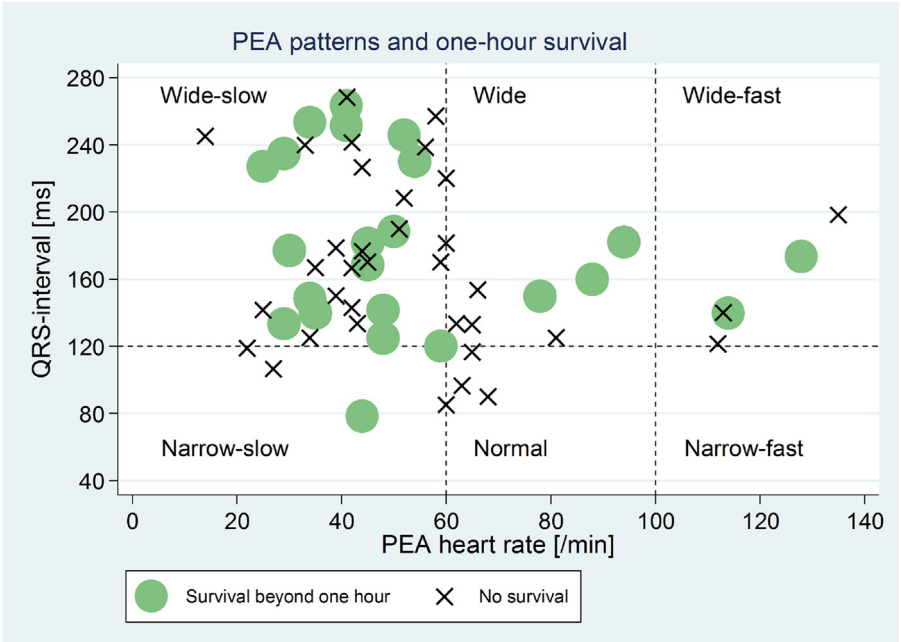

Fig. 4. 1-h survival from pulseless electrical activity (PEA) and patterns of early electrocardiogram (ECG), during resuscitation from in-hospital cardiac arrest. ROSC, return of spontaneous circulation.

adequate response to treatment of arrest and the underlying cause, or simply different timing of the ECG recordings.

There are several limitations to our study; utilizing data from only one hospital with a substantial amount of missing defibrillator files. This reduces both the internal and external validity. The reasons why defibrillator files were missing or lost were resuscitation of short duration and/or performed without defibrillator attached, exceeding memory capacity of local semi-automatic defibrillators, no file generated by the defibrillator despite proper functionality during ALS, files lost due to technical service, and erase of memory. With this low number of observations, the study may be considered as a pilot that needs to be confirmed.

The exact determination of QRS widths and QT intervals was challenging due to noise from one-channel ECG from defibrillator pads, which may have led to measuring errors. The normal-limits of ECG intervals and HR used this study were based on standard ECG normal values.

The strengths of this study are the prospective design, the thorough investigation of causes and the independent assessment of ECG intervals by two separate electrophysiologists.

Conclusion

In patients suffering in-hospital cardiac arrest where PEA was the first detected rhythm and a reliable cause of arrest was identified, the vast majority of episodes demonstrated abnormal ECG patterns in terms of widened QRS complexes and brady- or tachycardia. No cause-specific ECG pattern was found during the early phase of resuscitation, nor could survival be predicted based on the instantaneous ECG patterns defined in this study.

Conflict of interest statement

The authors Daniel Bergum, Trond Nordseth and Eirik Skogvoll have received research funding from the Norwegian Air Ambulance Foundation to accomplish the study. The Norwegian Air Ambulance Foundation had no influence on the study design, data collection, data analysis or drafting and revision of the manuscript.

None of the authors have any conflicts of interest to report.

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