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Clinical paper

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

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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

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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.