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Ischemic QRS Prolongation as a Predictor of Ventricular Fibrillation in a Canine Model

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Abstract

Objectives.—An acute coronary occlusion and its possible subsequent complications is one of the most common causes of death. One such complication is ventricular fibrillation (VF) due to myocardial ischemia. The severity of ischemia is related to the amount of coronary arterial collateral flow. In dog studies collateral flow has also been shown to be associated with QRS prolongation. The aim of this study was to investigate whether ischemic QRS prolongation (IQP) is associated with impending VF in an experimental acute ischemia dog model.

Methods.—Degree of IQP and occurrence of VF were measured in dogs (n=21) during coronary occlusion for 15 minutes and also during subsequent reperfusion (experiments conducted in 1984).

Results.—There was a significant difference in absolute IQP between dogs which developed VF during reperfusion (47 ± 29 ms, mean $\pm$ SD) and those which did not (12 ± 10 ms; $p=0.001$).

Conclusions.—IQP during acute coronary occlusion is associated with reperfusion VF in an experimental dog model and might therefore be a potential predictor of malignant arrhythmias in patients with acute coronary syndrome.

Keywords

Ventricular fibrillation; Ischemia; Electrocardiography; Acute coronary syndrome

Introduction

Acute myocardial infarction (AMI) is one of the most frequent causes of death in the western world [1]. Early diagnosis of an acute AMI is based on the presence of acute ischemia-induced ST-segment elevation (STEMI) or its equivalent ST depression on the presenting ECG [2], due to an acute coronary occlusion (ACO). The severity of the ischemia

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Disclosure of interest

The authors report no conflict of interest.

is related to both metabolic preconditioning and the amount of coronary arterial collateral flow [3,4]. Treatment of STEMI is aimed at accomplishing coronary reperfusion as soon as possible, either by percutaneous coronary intervention (PCI) or by intravenous thrombolytic therapy, in order to maximize myocardial salvage.[5] One serious complication of acute ischemia is ventricular fibrillation (VF). VF can occur both during ischemia and in conjunction with reperfusion therapy, although due to different pathophysiological mechanisms [6–11]. In both settings, however, these malignant arrhythmias can lead to sudden cardiac death (SCD) [8]. Identifying patients susceptible for VF would therefore be of great importance.

Severe ischemia-induced changes in the myocardium may not only be manifested by ST-segment deviation, but also by changes of the QRS complex [12–14]. Previous studies have documented the association between increased QRS duration during ischemia, decreased coronary collateral flow and rapid rate of myocardial cell death [3,15,16]. In a recent study we found ischemic QRS prolongation (IQP) to be a potential biomarker of severe myocardial ischemia [15], the method being recently validated by computer simulations [17]. Furthermore, transient ischemic QRS widening has been shown to predict impending VF and size of the myocardial injury in an experimental acute ischemia setting in pigs [18–20]. It has also been shown in humans that the magnitude of ST-segment elevation before PCI in STEMI independently predicts reperfusion VF [21].

The aim of this study was to test the hypothesis that IQP during the initial 15 minutes of acute coronary occlusion can serve as predictor for occurrence of VF within minutes following reperfusion in an experimental canine model.

Methods

Study Population

All experiments involving the use of laboratory animals conform to the guidelines of the American Physiological Society and the standards in the Guide for the Care and Use of Laboratory Animals, DHEW Publ. No. NIH 85–23, revised 1985, and was approved by the institutional review board.

Data from 21 healthy mongrel dogs, previously included in studies of reperfusion effects in late ischemic injury originally published in 1985, were included in the present study [22]. All dogs underwent proximal occlusion of the left circumflex coronary artery (LCX) for 15 minutes.

Surgical setup and ECG acquisition

All dogs were anesthetized with 30–40 mg/kg of sodium pentobarbital intravenously, intubated and ventilated as previously described in detail [23,22]. In short, a left thoracotomy was performed through the fourth intercostal space and the heart was suspended in a pericardial cradle. The LCX artery was identified and occluded for 15 minutes with a silk snare. Within the original experiment, investigating metabolic biomarkers in late ischemic injury, the dogs were divided into two groups, where the heart was excised at 3 or 20 minutes post-reperfusion. If the dog developed VF earlier than the

planned time for excision it was excised immediately following total cardiac arrest. Using a Gould model 2400 recorder, ECG lead II was recorded continuously before, during the occlusion and also during reperfusion until the heart was excised.

The ischemic bed size was estimated in nine dogs (no more data was available) from either visual inspection of the heart directly after occlusion or by injecting a fluorescent dye, that was inspected postmortem by one observer (RBJ) [22]. Occurrence of ischemia-induced cyanosis on the heart surface was recorded and graded as small or large in extent, providing an estimate of the extent of ischemic epicardium. Bed size grades were defined as either covering the apex of the heart (large) or not (small). The fluorescent dye thioflavine S (TS) (1.0 ml/kg of a freshly centrifuged 4% solution in normal saline), was injected intravenously 10–15 seconds before excision of the heart. After excision, the heart was cooled, dissected and put under ultraviolet light to distinguish between non-ischemic or mildly ischemic (TS fluorescent) and severely or totally ischemic (non-fluorescent) areas. Ischemia bed size grades were defined as above. Thioflavine S could not be used in dogs which developed VF.

ECG measurements

In acute STEMI, the terminal part of QRS complex is often distorted, without a clear demarcation between QRS and ST segment. Thus, in this setting it is often very difficult to determine the location of QRS offset. Therefore, Almer et al. developed a method to determine an estimate of the QRS offset, recently validated in computer simulations by Bacharova et al. [15,17] ECG measurements within this study were performed using this method.

In short, QRS waveform measurements were obtained from ECG lead II at a paper speed of 25mm/s and magnified x2 in a standard photocopier i.e. achieving 50mm/s and 20mm/mV. Before occlusion, measurement of a baseline QRS duration, defined as the time between QRS onset and the J-point, were undertaken in all animals. During occlusion IQP was measured at 1 minute intervals during first 5 minutes, the maximum value during this period was used in the data analysis.

IQP was measured by first recording the QRS duration using the J-point as offset if it could be clearly distinguished. When no J-point could be clearly distinguished due to ST elevation and terminal QRS distortion a line was drawn through the peak of the R (or R' if it was present) wave and along 40% of the downslope between the R peak and the nadir of the ST segment (Figure 1). The intersection of this line with the PR baseline was then used as offset. If there was an S wave associated with a ST-segment depression (basal lateral ischemia in LCX occlusions) a superimposed line from the S wave nadir along the first 40% of the S wave upslope was used and the intersection with the PR baseline marked the offset. Each value was measured as the average of measurements in 2 contiguous beats. The difference between the baseline QRS duration and the QRS duration during occlusion was calculated and referred to as absolute IQP (aIQP, measured to nearest 5 ms) and relative IQP (rIQP, normalized to baseline) (Figure 1). Out of the five measurements (at one minute intervals from occlusion), the measurement with the largest QRS duration, and thus aIQP, was used in the subsequent analysis. This QRS duration will be referred to as the max QRS

duration from here on. Furthermore, the timing of the max QRS duration was recorded to the nearest minute post occlusion.

Timing and occurrence of VF was recorded to the nearest second before or after reperfusion. VF was defined as occurrence of bizarre, irregular, random waveforms, no clearly identifiable QRS complexes or P waves and/or wandering baseline continuing for more than 10 seconds without reverting to sinus rhythm.

All waveform measurements were manually performed by one observer (JA). Results were adjudicated with an experienced observer (GW) if uncertainties arose.

Statistical analysis

Graphical procedures and comparisons of mean to median as well as the relation of the SD to the mean was used to evaluate if data were normally distributed. IQP was not normally distributed and part of most analyses. For parameters that was not normally distributed, non-parametric tests were applied for statistical significance. Continuous variables are presented as mean $\pm$ 1SD. Categorical data are presented as numbers. All statistical tests were 2-sided and a p value of <0.05 was considered to indicate statistical significance. A ROC analysis was performed to evaluate optimum cut-off value of IQP, using the Youden method. Sensitivity, specificity and area under the curve (AUC) was analyzed. SPSS version 19.0, SPSS version 23.0 and MATLAB version R2013a were used for the statistical analyses.

Results

The characteristics of the 21 dogs included in the study are shown in Table 1. Baseline QRS duration, max QRS duration and aIQP were 45 ± 4 , 77 ± 28 and 32 ± 28 ms, respectively. The maximum IQP was reached after 3.4 ± 1.0 minutes of occlusion. The IQP did not decrease from the maximum level, having reached a plateau, until reperfusion was accomplished at 15 minutes.

Twelve dogs (57%) developed VF after reperfusion, at 20 ± 11 s post-reperfusion. Mean aIQP was 47 ± 29 ms and differed significantly ($p=0.001$) from the 9 dogs that did not develop VF, 12 ± 10 ms (Table 1, Figure 2).

Of the 8 dogs in which ischemic bed size was recorded, 2 had a large bed size and 6 had a small bed size. None of the dogs with a small bed size developed VF. Out of the dogs with a large bed size one developed VF. There was no significant difference in mean aIQP between these groups.

The ROC analysis produced an optimum cut-off value of 21 ms with sensitivity of 92% and specificity of 89%. The AUC was 0.94 (95% CI 0.83 – 1.00)(Figure 4).

Time of excision, 3 or 20 minutes post-reperfusion, had no effect on the recorded results since all dogs which experienced VF developed their arrhythmia within 36 s after reperfusion.

Figure 3 shows example ECGs of two dogs, one which subsequently developed reperfusion VF and one which did not. The one which developed VF (top) also showed significant IQP.

Discussion

The present study shows a significant difference between dogs developing VF during reperfusion and those that did not, as regards acute IQP. Furthermore, in the included ROC analysis a AUC value of 0.94 was obtained with an optimum cut-off value of 21 ms with sensitivity of 92% and specificity of 89%. Thus, within the current study, predicting reperfusion VF already at 3.4 ± 1.0 minutes into occlusion is potentially possible.

Thus, an early QRS prolongation due to acute myocardial ischemia may have the potential to predict impending reperfusion VF, which is in accordance with the findings of Demidova et al. in an experimental pig model of myocardial infarction [18,19,21]. Earlier studies have also found that similar patterns to the ones found in our population positively predict VF in humans, although not appreciated as QRS prolongation [24]. Furthermore, IQP has been shown to correlate with collateral flow in a canine model [23,25,15]. Dogs with >5 ms of IQP exhibited significantly lower collateral flow compared to dogs with ≤ 5 ms IQP [15]. Hence, the increased risk of developing VF in acute ischemic heart disease might be related to more severe myocardial ischemia due to lower coronary collateral flow.

Translating the current results to a human population is, however, not necessarily straightforward. Within the study there were controlled experiments with total occlusion of the LCX in the dog. In a clinical setting this may differ widely where the initial thrombus may occlude the entire lumen of a vessel but also dissolve and/or add thrombus material continuously causing spontaneous reperfusion and/or reocclusion. While a spontaneous reperfusion in the acute phase of an AMI could be similar to the experimental setup, many more factors affect a clinical situation such as vessel spasms, previous heart disease etc.. Furthermore, Hedström et al. describes the difference in infarct evolution, using MRI, between dog and human, where the time to reach 50% of myocardium at risk is about 59% longer in humans than dogs [26]. Therefore, 15 minutes of occlusion probably corresponds to a longer time period in a human clinical setting. Although the current results have little implication in a clinical situation, it does, however, showcase IQP as a potential ECG marker.

Ischemic bed size was estimated in a subset within the current study. The results indicate that dogs with larger ischemic beds had longer QRS prolongation (albeit non-significant due to the coarse data recording). Also, none of the dogs with small bed size developed VF while one out of two in the large bed size group did.

In the experiment by Demidova et al. [18,20] myocardial infarction was induced through coronary artery occlusion lasting 40 minutes in an experimental pig model. Within this timeframe two separate peaks of QRS widening was observed. These peaks occurred at 3.7 ± 1.6 minutes and 19.1 ± 4.0 minutes into occlusion, the first similar in timing to the findings in the current study (3.7 ± 0.8 minutes). However, within the present study, the observers noted that the IQP developed into a plateau, after the maximum value had been

reached, until reperfusion. This is dissimilar to the findings by Demidova et al where the QRS widening peaked in order to reduce before the next peak. There are distinct differences between the experimental models, however, foremost that of different animals used (dog versus pig) that can explain the discrepancies but also obstruct the translation of results. Furthermore, the current study was designed to study ischemia only, and therefore duration of ischemia was set to 15 minutes in order to avoid infarct development. In the study by Demidova et al the duration of ischemia was 40 minutes in order for the animals to develop infarction [18]. It might be that biphasic QRS prolongation reflects both the initial ischemia and the start of infarct development at approximately 20 minutes.

Within the present study, ST-segment elevation was not considered since only one lead was available and no global ST-segment elevation could be appreciated. However, Weston et al. reported in a dog model that for a given magnitude of STE, the presence of concurrent QRS prolongation was associated with less myocardial salvage [25]. Furthermore, Demidova et al. found that the magnitude of ST-segment elevation before PCI for STEMI independently predicts reperfusion VF [21]. Thus, evaluating ST-segment elevation together with IQP in patients with STEMI might provide additional diagnostic information.

Limitations

The current study should be viewed in the light of some limitations. First, an animal model with small sample size was used. To what extent QRS prolongation can be used to predict AMI patients at risk of malignant arrhythmias is not known. Second, occlusion of only the LCX was investigated. Furthermore, only one ECG lead (lead II) was recorded and assessed. Different results may have been obtained if additional arteries and/or leads were assessed. However, within this study there are no means of optimizing the experiment setup more than 30 years later. Third, IQP measurements were made manually. Development of automated or semi-automated methods for IQP measurements would be desirable and necessary for clinical feasibility. Also a 12-lead ECG-method for quantifying ischemia-induced QRS prolongation would be paramount and has actually been presented in a recent study by Elmberg et al. based on the current method [27]. Fourth, in the current study only the first 15 minutes of coronary occlusion and a various degree of ischemia were evaluated. Studies in STEMI patients regarding development and temporal evolution of QRS prolongation during the first hours of ACO as well as its correlation with myocardial salvage are needed in order to elucidate whether the findings in the present study are similar to findings in a clinical setting in humans. Fifth, ischemic bed size was estimated within the current study based on visual inspection of cyanosis on the heart surface. The coarseness of the grading is a major limitation and the data should be viewed in the light of this.

Conclusion

There was a significant difference in IQP between dogs developing VF after reperfusion of an ACO as compared to those who did not. Thus, an early QRS prolongation due to acute myocardial ischemia may have the potential to predict impending reperfusion VF.

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Figure 1. Depiction of ischemic QRS prolongation measurement method

Depiction of the ischemic QRS prolongation measurement method. During ischemia when no J-point could be clearly distinguished due to ST elevation, a line was drawn through the peak of the R (or R' if it was present) wave and along 40% of the downslope between the R peak and the nadir of the ST segment. Reprinted from Journal of Electrocardiology, 49/2, Almer *et al.*, Ischemic QRS prolongation as a biomarker of severe myocardial ischemia, 139-147, Copyright (2016), with permission from Elsevier.

Figure 2. Relation between VF groups.
Box plot of reperfusion VF and no VF groups.

Figure 3. Example ECGs

Example ECG of lead II in two dogs before and at 1, 2, 3 and 4 minutes into occlusion. The dog at the top later develops VF while the one at the bottom does not. The one at the top develops ischemic QRS prolongation during the first minutes of occlusion.

Figure 4. ROC curve
ROC curve of IQP indicating VF or not.

Table 1.

Baseline characteristics

Mean $\pm$ 1SD (range)	Total (n=21),	VF (n=12)	No VF (n=9)	P-value [*]
Pre-occlusion heart rate (bpm)	166 $\pm$ 31 (124-260)	170 $\pm$ 33 (130-260)	160 $\pm$ 29 (124-222)	0.434
Pre-occlusion blood pressure systolic/ diastolic (mmHg)	166 $\pm$ 29 (120-230) / 111 $\pm$ 2 (85-190)	159 $\pm$ 22 (120-190) / 114 $\pm$ 29 (85-190)	174 $\pm$ 35 (140-230) / 108 $\pm$ 17 (90-130)	0.336 / 0.775
Pre-occlusion QRS duration (ms)	45 $\pm$ 4 (35-53)	44 $\pm$ 3 (35-48)	47 $\pm$ 4 (42-53)	0.137
Max QRS duration (ms)	77 $\pm$ 28 (42-155)	91 $\pm$ 28 (50-155)	59 $\pm$ 10 (42-73)	0.004
Absolute IQP (ms)	32 $\pm$ 28 (0-115)	47 $\pm$ 29 (5-115)	12 $\pm$ 10 (0-23)	0.001
Relative IQP (%)	73 $\pm$ 68 (0-288)	108 $\pm$ 70 (11-288)	26 $\pm$ 19 (0-45)	0.001
Time to max IQP (minutes)	3.4 $\pm$ 1.0 (2.0-5.0)	3.7 $\pm$ 0.8 (3.0-5.0)	3.0 $\pm$ 1.0 (2.0-4.0)	0.181

IQP: Ischemic QRS prolongation

*
Mann-Whitney test