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EXPERIMENTAL PAPER

Influence of the skeletal muscle activity on time and frequency domain properties of the body surface ECG during evolving ventricular fibrillation in the pig☆

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Summary
Aim of the study: To evaluate influence of the skeletal muscle activity (SMA) on time and frequency domain properties of ECG during VF.
Materials and methods: We studied the first 9 min of electrically induced VF ($N = 7$). We recorded Lead II ECG, 247 unipolar epicardial ventricular electrograms (UEGs) and 3 bipolar skeletal electromyograms (EMGs) near the positions of the ECG electrodes (sampling rate, 500 Hz). We reconstructed ECG (RECG) from UEGs using forward-solution transformation matrix. Spectral properties of ECG, RECG, UEGs and MEGs were assessed in the range 2—250 Hz by the median frequency (MF) and the upper limit of frequency range containing 99% of spectral energy ($F_{lim}(99)$). Scaling exponent of ECG, RECG and EMGs was calculated in the ranges of 1—8 and 5—20 sampling intervals (ScE_{1-8} and ScE_{5-20}, respectively).
Results: We observed non-monotonic increases in MF and $F_{lim}(99)$ of the ECG, but not UEGs and RECG, at 1—5 min of VF. Maximum values of MF and $F_{lim}(99)$ in ECG, UEGs and RECG were (in Hz): 32 ± 29 and 166 ± 67; 11 ± 2 and 36 ± 7; 10 ± 2 and 32 ± 6, respectively. The transient increases in the high-frequency content of the ECG were correlated with enhanced activity in EMGs, characterized by an almost uniform spectrum in the range 2—250 Hz ($MF = 92 \pm 29$; $F_{lim}(99) = 245 \pm 4$ Hz). Peak values of ScE_{1-8} were the highest in EMGs (1.95 ± 0.04), intermediate in the ECG (1.59 ± 0.26), and the lowest in RECG (1.088 ± 0.007).
Conclusion: SMA significantly contributes to ECG during VF and can bias metrics used for assessment of VF organization.
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Introduction

The body surface ECG is commonly the only source of information available to a health care professional at the scene of out-of-hospital ventricular fibrillation (VF). Many attempts have been made to use the measures based on the ECG waveform in frequency and time domain as a means of assessing VF duration for optimization of the resuscitation strategies.[1–11] Accumulated clinical and laboratory data[1–11] have shown that these ECG waveform-based measures, or combinations of these measures, can help to predict the likelihood of the successful countershock therapy in cardiac arrest patients. However, analysis of ECG during VF can be confounded by extra-cardiac phenomena. Knowledge about sources of extra-cardiac signals and estimation of their contributions into the ECG signal are important for interpretation of the ECG recordings and hence for understanding how the surface ECG is related to cardiac electric activity during VF.

SMA is a significant source of bioelectrical activity which contaminates ECG. However, whereas the contribution of SMA is well studied for the case of regular cardiac rhythms in which the constituent waves of the ECG can be clearly distinguished from SMA, potential contribution of SMA to the ECG during VF has not been addressed. Thus, the purpose of this study was to investigate dynamics of the SMA and to evaluate its influence on time and frequency domain properties of body surface ECG during evolving VF in a swine model.

Materials and methods

Seven pigs (22.8 ± 4.0 kg) were sedated with acepromazine (0.4 mg/kg), intubated and ventilated either with room air or isofluorane in oxygen depending on the type of anesthesia. In all animals, oxygen saturation was continuously monitored with a pulse oximeter and arterial blood samples were taken prior to induction of VF. Parameters of ventilation were adjusted to achieve oxygen saturation of at least 95%, pO_2 above 80 Hg, and pCO_2 between 35 and 45 mmHg. Details of the experiments including the type of anesthesia are summarized in Table 1. In five experiments, the chest was opened via median sternotomy. In 4 open-chest pigs, Lead II ECG, 3 EMGs and 247 epicardial UEGs were recorded. EMGs were recorded with 3 silver bipolar electrodes (interpolar distance = 0.5 mm) inserted into skeletal muscles adjacent to the positions of ECG leads. UEGs were recorded using a sock electrode with 247 unipolar leads uniformly distributed over the epicardial surface of the ventricles.[12] In one open-chest pig the heart was isolated and perfused with blood in the Langendorff apparatus.[13] A volume-conductor ECG was recorded using two silver electrodes and a ground electrode immersed in the superfusion chamber.[13] In 2 closed-chest pigs, only Lead II ECG and 3 EMGs were recorded. All signals were passed through a wide band-pass AC-coupled preamplifier and digitized at 500 Hz. In all animals VF was electrically induced and measurements were taken from 0 to 9 min of VF. Animal protocols were approved by the Institutional Animal Care and Use Committee of the University of Utah.

Using information about the 3-dimensional geometry of the sock electrode including the positions of the individual UEG leads and a realistic geometry of the pig torso, we generated a forward-solution transformation matrix[14–16] which was used for the reconstruction of the ECG (RECG) which approximated Lead II configuration used in the experiment.

Time domain properties of UEGs, ECGs, RECGs and EMGs were characterized in terms of fractal self-similarity dimension estimated by scaling exponent (ScE). This measure shows how the signal under consideration differs from the random one, which is characterized by ScE ≈ 2.[6] We estimated ScE as described previously.[1,3] Briefly, we calculated function $L(k)$ for lag $k = 1-1024$.

$$L(k) = \frac{2048}{k(2048-k)} \sum_{i=1}^{2048-k} \left| X(i) - X(i+k) \right|$$

where $X(i)$ is the i th measurement of the signal waveform. The resulting $L(k)$ was fitted to the following exponential function $L_f(k)$ using log–log representation of $L(k)$ and $L_f(k)$ versus k:

$$L_f(k) = k^{(1-\mathrm{ScE})}$$

The value of ScE was obtained as the result of this fitting procedure. It should be noted that the value of ScE may depend on fitting interval of k. This interval varied in previous studies.[1,3,17]. We used two different fitting intervals

Table 1 Summary of the experiments

Experiment #	ECG	EMG	UEG	Conditions	Anesthesia
Exp #1	Yes	Yes	Yes	Opened chest	Isofluorane, 1–2% in oxygen
Exp #2	Yes	Yes	Yes	Opened chest	Isofluorane, 1–2% in oxygen
Exp #3	Yes	Yes	Yes	Opened chest	Isofluorane, 1–2% in oxygen
Exp #4	Yes	No	Yes	Opened chest	Pentobarbital, 40 mg/kg, I.V. Ventilation with room air
Exp #5	Yes	Yes	No	Closed chest	α-Chloralose (40 mg/kg loading dose; 10 mg/kg per hour thereafter). Ventilation with room air
Exp #6	Yes	Yes	No	Closed chest	Isofluorane, 1–2% in oxygen
Exp #7	Volume-conducted ECG	No	No	Langendorff-perfused with blood	Pentobarbital, 40 mg/kg, I.V. Ventillation with 95% O_2/5% CO_2