Published in final edited form as:

Resuscitation. 2008 August ; 78(2): 215–223. doi:10.1016/j.resuscitation.2008.03.010.

Influence of the Skeletal Muscle Activity on Time and Frequency Domain Properties of the Body Surface ECG during Evolving Ventricular Fibrillation in the Pig

Alexander G. Shvedko, Mark D. Warren, Shibaji Shome, Jeroen Stinstra, and Alexey V. Zaitsev
Nora Eccles Harrison Cardiovascular Research and Training Institute, University of Utah, Salt Lake City, UT

Abstract

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 minutes 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, 500Hz). We reconstructed ECG (RECG) from UEGs using forward-solution transformation matrix. Spectral properties of ECG, RECG, UEGs and MEGs were assessed in the range 2–250Hz 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 minutes 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 ± 29 ; $F_{lim}(99)$ = 245 ± 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.

Keywords

electrocardiography; ventricular fibrillation; asystole; ischemia; fast Fourier transformation

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

Corresponding author: Alexander G. Shvedko, PhD Nora Eccles Harrison CVRTI University of Utah 95 South 2000 East Salt Lake City, UT 84112–5000 Phone (801) 585–1090 Fax (801) 581–3128 E-mail shvedko@cvrti.utah.edu.

Conflict of interest There is no conflict of interest.

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%, pO₂ above 80 Hg, and pCO₂ between 35 and 45 mmHg. Details of the experiments including the type of anesthesia are summarized in Table 1. In 5 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 minutes 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 [14,15,16] 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 $k = 1-1024$.

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

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 :

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

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 (1 to 8 and 5 to 20 points) for calculation of ScE and designated them as ScE_{1-8} and ScE_{5-20} , respectively.

In the frequency domain we analyzed two metrics. Median frequency (MF) of the power spectrum is the frequency for which the integrated signal power was one half the total integrated power from 2 to 250 Hz [6,11]. We also computed function $F_{lim}(p)$ which estimated the upper limit of the frequency range containing p percent of the total integrated power in the range from 2 to 250 Hz. In particular, $F_{lim}(99)$ is a useful indicator of how far the frequency content of a signal extends to the frequency limit determined by the sampling rate (250 Hz in our case). All calculations were done for 4-second epochs (2048 samples) taken at 0–9 min of VF.

Results

In all whole animal experiments we observed non-monotonic increases of electrical activity in high frequency range of the ECG spectrum within the first 5 minutes of VF. An example (exp #5, see Table 1) is shown in Figures 1 and 2. The presence of high-frequency activity in the ECG corresponded to transient increases in the amplitude and the total spectral power of at least one EMG signal (see Figs. 1 and 2).

Surface plot in Figure 3 shows F_{lim} measured for ECG, RECG and an EMG in exp#1 as the function of p (percent of total power) and t (VF duration). Note that the shapes of the $F_{lim}(p,t)$ surfaces constructed for ECG and RECG are similar up to the level of $p \approx 89\%$, but are dissimilar in the range of p between 90% and 99%. Specifically, $F_{lim}(99)$ of RECG is contained within ~ 40 Hz throughout 9 min of VF, whereas $F_{lim}(99)$ of ECG extends to higher frequencies, reaching a peak of 198 Hz at 3 min of VF. This peak corresponds to a transient increase of EMG activity similar to that shown in Fig. 1. In contrast to ECG and RECG, $F_{lim}(p)$ of EMG is almost linear throughout the first 9 min of VF. This indicates that the spectrum of EMG is almost uniform in the range studied (2–250 Hz).

Figure 4 shows $F_{lim}(99)$ and MF for ECG, RECG, EMG and UEG in all experiments where the specific experimental conditions allowed to record particular signals. One can see that $F_{lim}(99)$ of ECG changes non-monotonically over the time course of VF and is highly variable among experiments (Fig. 4A). The time course of ECG MF is also different between experiments (Fig. 4B). Note that in experiments #4 and #5 the peak of ECG MF reaches 48 and 90 Hz, respectively, which corresponds to the time points when $F_{lim}(99)$ reaches peak values in those experiments. Figure 4 C and D shows $F_{lim}(99)$ and MF for EMG signals. In all experiments, $F_{lim}(99)$ of EMG was very close to 250 Hz (Fig. 4C), and MF of EMG was in the range of 60–130 Hz (Fig. 4, C and D). This reflects relatively uniform distribution of the spectral power of EMG over the range 2–250 Hz, albeit slightly skewed towards lower frequencies. In contrast to ECG, both epicardial UEGs and RECG did not have high frequency components throughout the first 9 minutes of VF. In particular, MF and $F_{lim}(99)$ of RECG never exceeded 15 and 40 Hz, respectively (see Fig. 4, E-F). The average values of $F_{lim}(99)$ and MF computed for UEGs were directly correlated to those of RECG throughout the first 9 min of VF ($R^2=0.76$ and 0.96 , respectively). In contrast, the correlation between $F_{lim}(99)$ and MF computed for UEGs and those parameters computed for true ECG was very poor ($R^2=0.11$ and 0.01 , respectively).

Given the potential utility of ScE as the measure of VF organization (see Discussion), we investigated the dynamics of ScE calculated for ECG, MEG, UEGs and RECG. ScE was computed using two different intervals of k (ScE_{1-8} and ScE_{5-20} , see Methods). The time course of ScE of ECG was highly variable among experiments, it was non-monotonic in the

majority of experiments, and it was different for ScE_{1-8} and ScE_{5-20} (see Fig. 5, A and B). Note that the peak values of ScE reached in experiments # 4 and #5 at minute 1 and minute 2 of VF, respectively (see Fig. 5, A and B), coincided with surges of high-frequency activity reflected by sharp increases in $F_{lim}(99)$ in these experiments (see Fig. 4 A and B). Overall, there was a direct correlation between $F_{lim}(99)$ and ScE_{1-8} of ECG ($R^2=0.86$) indicating that ScE_{1-8} is highly sensitive to high-frequency components of ECG.

Both ScE_{1-8} and ScE_{5-20} measured for EMGs were close to 2 throughout the first 9 min of VF, which is consistent with a broad, white noise-like spectrum of EMGs shown in Fig. 2. The dynamics of RECG ScE depended on the choice of fitting interval. ScE_{1-8} was close to 1 throughout the first 9 min of VF (Fig. 5E), whereas ScE_{5-20} had higher values and a non-monotonic dynamics which somewhat followed the dynamics of MF and $F_{lim}(99)$ computed for UEGs and RECG (see Fig. 4,E-H).

Since the value of ScE is sensitive to the choice of the fitting interval of k , we examined qualitatively the dynamics of the entire L-curve. Figure 6 shows the selected representative cases of L-curve computed for ECG from open-chest animal (Fig. 6A), RECG from the same animal (Fig. 6B), ECG from a closed-chest animal (Fig. 6C) and volume-conducted ECG from an isolated heart (Fig. 6D). Steep slopes could be seen only in L-curves computed for whole animal ECGs (arrowheads in Fig. 6 A and C). However, L-curves computed for RECG (Fig. 6B) and the volume-conducted ECG of the isolated heart (Fig. 6D) maintained shallow plateau throughout the first 9 min of VF.

Table 2 highlights the differences between maximum values of $F_{lim}(99)$, MF, and ScE_{1-8} measured for ECG, EMGs, RECG, and UEGs during the first 9 minutes of VF.

Discussion

Electrical activity of skeletal muscles during VF

We are not aware of any prior investigation concerning the presence of skeletal myopotentials during VF and their effect on the body surface ECG. We found that, both in closed and opened-chest pigs, marked increases in SMA can occur after the onset of VF. In our study EMG signals exhibited a very broad spectrum in the measured frequency band (2–250 Hz), which resembles muscular “fibrillation” resulting from asynchronous firing of the individual fibers of a motor unit following denervation [18,19]. Speculatively, the mechanism of SMA observed in our study may involve hyperexcitability in the motor neurons and/or skeletal muscle fibers, resulting from extracellular accumulation of potassium and depolarization of the membrane potential [20,21].

It is unknown what governs the dynamics of SMA after the onset of VF. In animal studies, it may depend on various experimental conditions, such as the type of anesthesia and use of paralytics. We predict that differences in SMA may significantly affect the time course of the ScE during VF. Indeed, the time course of ScE appears to be different in animals anesthetized with alpha-chloralose and subjected to muscle paralysis (see Fig. 2 in Ref. 17) and those with isoflurane anesthesia and no paralytics used (see Fig. 6A in Ref. 3). In any case, we should note that, in the setting of out-of-hospital cardiac arrest, neither anesthesia nor chemical paralysis is expected, and therefore the time course of SMA in patients may be different from that observed in the relevant animal studies.

Spectral properties of cardiac signal during VF

To the best of our knowledge, this is the first study to compare the spectral properties of epicardial UEGs, forward-solution RECG, and real body surface ECG during progression of VF. In our study, the MF and $F_{lim}(99)$ of UEGs were highly correlated with those of RECG,

with MF and $F_{lim}(99)$ not exceeding 15 and 40 Hz, respectively. This agrees with previous findings of compactness and good correlation between the spectral content of optical action potentials and volume-conducted ECG in isolated, globally ischemic porcine hearts [13]. Since the excitation frequency in human VF is lower than in porcine VF [22], the upper limit of frequency content of cardiac activity during VF in patients is expected to be lower than that in pigs. Therefore, for quantitative analysis of VF ECG, it seems to be advisable to use a relatively aggressive low-pass filtering. However, further studies are required to develop more definitive guidelines for such filtering.

Is ScE a reliable measure of VF organization?

ScE was recently introduced as a measure of ECG organization during VF [1-9,17]. It was postulated that ScE distinguishes “coarse” VF (low value of ScE close to 1) from “fine” VF (high value of ScE close to 2) and can provide a means to estimate VF duration [2] and to predict the outcome of rescue shocks [1]. It was hypothesized that ScE reflects spatiotemporal organization of myocardial depolarization during VF [1] but this conjecture has never been substantiated. The value of ScE depends on the determination of the range of time scales over which ECG presumably exhibits scaling properties. Unfortunately, any rigorous criteria for the choice of this range are lacking and it varied substantially in previous studies [1,3,17]. It should be noted however that the value of ScE is very sensitive to the choice of the fitting interval. For example, in case of RECG, we observed higher values of ScE_{5-20} as compared to ScE_{1-8} (see Fig. 6, E and F). However, this merely reflected the fact that fitting interval $k=5-20$ extended slightly beyond the plateau region of the L-curve. This caused an increase in the negative slope of the fitting line, which no longer fitted a linear region.

A more important problem, however, is correct understanding of factors shaping the L-curve. The ranges of k previously used for computing ScE in porcine model of VF corresponded (collectively) to frequency range 25–200 Hz [1,3,17]. We argue that in this frequency range the representation of cardiac VF waves is minor and is overshadowed by extracardiac sources including SMA. The qualitative relationship between L-curve and low- and high- ranges of the ECG spectrum is illustrated in Fig. 8. Fig. 8A shows L-function computed for original ECG signal (exp #4). Fig. 8, B and C show L-function computed for time sequences corresponding to low (< 15 Hz) and high (> 15 Hz) frequency components of the original ECG spectrum. The choice of the cutoff frequency is somewhat arbitrary but it is based on the perceived boundary between the compact part of the spectrum and a flat “floor” extending to the frequency limit. It is clearly seen that removal of low-frequency component of the ECG (mostly representing the cardiac signal) does not change much the shape of the L-curve (Fig. 8H), whereas removal of high-frequency component creates a prominent plateau in the L-curve (Fig. 8E) with a slope close to 0 (ScE close to 1). This kind of plateau was consistently observed in L-curves computed for RECG or volume-conducted ECG, i.e. surrogate ECG signals in which contamination from extracardiac sources is minimal (see Fig. 6, B and D). This indicates that VF does not reach random state throughout the first 9 minutes and strongly suggests that steep L-curves found in the case of real ECG (see arrows in Fig. 8, A and C) are due to non-cardiac sources.

In general, the value of ECG ScE probably depends on the relative contribution of cardiac signal with compact spectrum and extracardiac (including SMA) signal with a broad (white noise-like) spectrum. Accordingly, ScE may increase either when the power of cardiac component decreases amid a constant power of noise-like signal, or when the power of noise-like component increases amid a constant power of cardiac signal. The total power of ECG usually decreases steadily over time of VF [6]. Therefore, with constant contribution of noise-like signal, ScE should increase monotonically, culminating in $ScE \approx 2$ during asystole [9]. This is in agreement with the strong correlation found between ScE and ECG root-mean-square

amplitude in human VF ECG data [1]. However, our data indicate that, if a non-monotonic increase in SMA occurs, ScE value may also change non-monotonically with time of VF.

Conclusion

SMA may significantly contribute to the body surface ECG during porcine VF. True cardiac activity during VF has a compact power spectrum below 40 Hz, whereas SMA has a broad power spectrum extending to at least 250 Hz. Caution should be exercised in interpretation of ECG ScE during VF, since increased values of ScE may reflect increased contribution of SMA and other extra-cardiac sources. As such, critically high values of ScE (approaching 2) are perhaps a reliable indicator of asystole, but intermediate values of ScE are more difficult to interpret in terms of underlying VF organization.

Acknowledgements

Superb technical assistance of Ms. Jayne Davis and Ms. Alicja Booth is greatly appreciated. This study was supported by a research grant from Nora Eccles Treadwell Foundation and NIH (1R01HL088444-01A1). The sponsors were not involved in any way in the preparation of this manuscript.

References

1. Callaway CW, Sherman LD, Mosesso VN, Dietrich TJ, Holt E, Clarkson MC. Scaling Exponent Predicts Defibrillation Success for Out-of-Hospital Ventricular Fibrillation Cardiac Arrest. *Circulation* 2001;103:1656–1661. [PubMed: 11273993]
2. Menegazzi JJ, Callaway CW, Sherman LD, Hostler DP, Wang HE, Fertig KC, Logue ES. Ventricular Fibrillation Scaling Exponent Can Guide Timing of Defibrillation and Other Therapies. *Circulation* 2004;109:926–931. [PubMed: 14757695]
3. Callaway CW, Sherman LD, Scheatzle MD, Menegazzi JJ. Scaling Structure of Electrocardiographic Waveform During Prolonged Ventricular Fibrillation in Swine. *PACE* 2000;23:180–191. [PubMed: 10709226]
4. Sherman LD, Callaway CW, Menegazzi JJ. Ventricular fibrillation exhibits dynamical properties and self-similarity. *Resuscitation* 2000;47:163–173. [PubMed: 11008154]
5. Menegazzi JJ, Wang HE, Lightfoot CB, Fertig KC, Chengelis NL, Sherman LD, Callaway CW. Immediate defibrillation versus interventions first in a swine model of prolonged ventricular fibrillation. *Resuscitation* 2003;59:261–270. [PubMed: 14625118]
6. Callaway CW, Menegazzi JJ. Waveform analysis of ventricular fibrillation to predict defibrillation. *Cardiopulmonary Resuscitation* 2005;11:192–199.
7. Wang HE, Menegazzi JJ, Lightfoot CB, Callaway CW, Fertig KC, Sherman LD, Hsieh M. Effects of biphasic vs monophasic defibrillation on the scaling exponent in a swine model of prolonged ventricular fibrillation. *Academic Emergency Medicine* 2001;8:771–780. [PubMed: 11483451]
8. Angelos MG, Menegazzi JJ, Callaway CW. Bench to Bedside: Resuscitation from Prolonged Ventricular Fibrillation. *Academic Emergency Medicine* 2001;8:909–924. [PubMed: 11535487]
9. Lightfoot CB, Sorensen TJ, Garfinkel MD, Sherman LD, Callaway CW, Menegazzi JJ. Physician interpretation and quantitative measures of electrocardiographic ventricular fibrillation waveform. *Prehospital Emergency Care* 2001;5:147–154. [PubMed: 11339724]
10. Makikallio TH, Huikuri HV, Myerburg RJ, Seppanen T, Kloosterman M, Interian A, Castellanos A, Mitrani RD. Differences in the activation patterns between sustained and self-terminating episodes of human ventricular fibrillation. *Annals of Medicine* 2002;34:130–135. [PubMed: 12108576]
11. Strommenger HU, Lindner KH, Brown CG. Analysis of the Ventricular Fibrillation ECG Signal Amplitude and Frequency Parameters as Predictors of Countershock Success in Humans. *Chest* 1997;111:584–589. [PubMed: 9118691]
12. Arisi G, Macchi E, Corradi C, Lux RL, Taccardi B. Epicardial excitation during ventricular pacing. Relative independence of breakthrough sites from excitation sequence in canine right ventricle. *Circ. Res* 1992;71:840–849. [PubMed: 1381295]

13. Huizar JF, Warren MD, Shvedko AG, Kalifa J, Moreno J, Mironov SF, Jalife J, Zaitsev AV. Three Distinct Phases of VF during Global Ischemia in the Isolated Blood-Perfused Pig Heart. *Am J Physiol Heart Circ Physiol* 2007;293:1617–1628.
14. Barr RC, Pilkington TC, Boineau JP, Spach MS. Comparing methods of generating surface potentials. *IEEE Trans Biomed Eng* 1966;13:210–211. [PubMed: 5978295]
15. de Munck JC. A linear discretization of the volume conductor boundary integral equation using analytically integrated elements. *IEEE Trans Biomed Eng* 1992;39:986–990. [PubMed: 1473829]
16. Ferguson AS, Stroink G. Factors affecting the accuracy of the boundary element method in the forward problem - I: Calculating surface potentials. *IEEE Trans Biomed Eng* 1997;44:1139–1155. [PubMed: 9353994]
17. Lightfoot CB, Nremt P, Callaway CW, Hsieh M, Fertig KC, Sherman LD, Menegazzi JJ. Dynamic nature of electrocardiographic waveform predicts rescue shock outcome in porcine ventricular fibrillation. *Ann Emerg Med* 2003;42:230–241. [PubMed: 12883511]
18. Salafsky B, Bell J, Prewitt MA. Development of fibrillation potentials in denervated fast and slow skeletal muscle. *Am J Physiol* 1968;215:637–643. [PubMed: 5671001]
19. Keller SP, Sandrock AW, Gozani SN. Noninvasive detection of fibrillation potentials in skeletal muscle. *IEEE Trans Biomed Eng* 2002;49:788–795. [PubMed: 12148817]
20. Bostock H, Baker M, Grafe P, Reid G. Changes in excitability and accommodation of human motor axons following brief periods of ischemia. *J Physiol* 1991;441:513–535. [PubMed: 1816385]
21. Bostock H, Baker M, Reid G. Changes in excitability of human motor axons underlying post-ischemic fasciculations: evidence for two stable states. *J Physiol* 1991;441:537–557. [PubMed: 1667800]
22. Martin DR, Brown CG, Dzwonczyk R. Frequency analysis of the human and swine electrocardiogram during ventricular fibrillation. *Resuscitation* 1991;22:85–91. [PubMed: 1658897]

Figure 1.
ECG (A,B,C) and EMG (D,E,F) during VF in a closed chest animal. *Top to bottom*, results obtained at 0, 1 and 5 minutes of VF.

Figure 2.
Amplitude spectra corresponding to the time sequences presented in Figure 1.

Figure 3.

Surface plots of F_{lim} versus time of VF and P (percent of total power). **A, C and E** results for ECG, RECG and EMG, respectively. **B, D and F** the same but with the range of F_{lim} from 2 to 40 Hz expanded.

Figure 4. Spectral parameters plotted versus time of VF. *Left, $F_{lim}(99)$. Right, MF. Top to bottom, results obtained for ECG, EMG, RECG and UEGs.*

Figure 5.

Scaling exponent (ScE) plotted versus time of VF. *Left*, ScE computed using fitting interval of lag $k = 1-8$ (ScE_{1-8}). *Right*, ScE computed using fitting interval of lag $k = 5-20$ (ScE_{5-20}). *Top to bottom*, results obtained for ECG, EMG and RECG.

Figure 6.

Surface plots of L -function versus time lag (k) and time of VF (min). $L(k)$ is plotted in log-log scale and normalized to the maximum value in each experiment. The steepest L -curves corresponding to highest values of ScE are shown with arrows. Plots are presented for the following data: **A**, ECG from an open-chest animal (Exp #1); **B**, RECG from an opened-chest animal (Exp #1); **C**, ECG from a closed-chest animal (Exp #5); **D**, volume-conducted ECG from an isolated blood-perfused heart (Exp #7).

Figure 7.

A qualitative illustration of the relationship between low- (< 15 Hz) and high- (> 15 Hz) frequency components of body surface ECG and L -function. ECG data from exp #4. *Left to right*, ECG, corresponding L -function plotted in log-log scale, and the ECG spectrum. *Top to bottom*, original ECG signal (0–250 Hz), low-frequency (0–15 Hz) component of ECG, and high-frequency (15–250 Hz) component of ECG.

Table 1

Summary of the experiments

Experiment #	ECG	EMG	UEG	Conditions	Anesthesia
Exp #1	Yes	Yes	Yes	Opened chest	Isoflurane, 1–2% in oxygen
Exp #2	Yes	Yes	Yes	Opened chest	Isoflurane, 1–2% in oxygen
Exp #3	Yes	Yes	Yes	Opened chest	Isoflurane, 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	Isoflurane, 1–2% in oxygen
Exp #7	Volume-conducted ECG	No	No	Langendorff-perfused with blood	Pentobarbital, 40 mg/kg, I.V. Ventilation with 95% O ₂ /5% CO ₂

Table 2Maximal values of $F_{lim}(99)$, MF and ScE_{1-8} during the first 9 min of VF averaged among different experiments

Type of electrogram	N	Max $F_{lim}(99)$, Hz	Max MF, Hz	Max ScE_{1-8}
UEG	4	35.6±6.3	10.8±1.8	-
RECG	4	32.2±5.5	10.1±1.7	1.088±0.007
ECG	6	165.9±66.9	31.9±29.2	1.59±0.26
EMG	5	245.3±3.9	91.5±28.8	1.95±0.04