The duration of ventricular fibrillation required to produce pulseless electrical activity

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Abstract The duration of untreated (no cardiopulmonary resuscitation) ventricular fibrillation (VF) needed to produce postdefibrillation pulseless electrical activity (PEA) was determined in 9 anesthetized swine ranging in weight from 20 to 30 kg. VF was induced electrically by a right ventricular catheter electrode, while arterial pressure and the electrocardiogram were recorded. VF was confirmed by the presence of VF waves in the electrocardiogram and a loss of pulsatile arterial pressure. VF was allowed to persist for 15-second increments (eg, 15, 30, 45, etc), after which defibrillation was achieved with transthoracic electrodes and the presence or absence of PEA was noted. If PEA was present, rhythmic chest compressions were applied to rescue the animal. Just after initiation of VF and just before defibrillation, VF wave frequency was measured. PEA was encountered in 100\% of the trials after 180 seconds of VF. The threshold duration for PEA was 60 seconds. VF wave frequency decreased with the passage of time. At VF initiation, VF wave frequency ($f_0$) ranged from 6 to 15 per second, with a mean of 10.1 $\pm$ 2.1 per second. At 180 seconds ($f_{180}$), the mean frequency was 4.0 $\pm$ 0 per second. It was only possible to eliminate PEA and restore pumping in 1 animal when untreated VF lasted more than 180 seconds. There was no clear transition in the frequency of the VF waves with the passage of time that could predict the possibility of postdefibrillation PEA. Moreover, because of the different initial VF wave frequencies and the different rates of decrease with time, a measurement of VF wave frequency is unlikely to be informative on how long VF had been present. A consistent finding in this swine study of prolonged untreated VF was a rise in blood K\textsuperscript{+} which increased from a normal prefibrillation value of about 4 mEq/L to 8 to 12 mEq/L at 180 seconds. The longer the duration of VF, the higher the K\textsuperscript{+}.

1. Introduction

Pulseless electrical activity (PEA), formerly called electromechanical dissociation, is a postventricular defibrillation condition in which the R waves of the electrocardiogram (ECG) are present but are not followed by ventricular contractions. It likely underlies the reason for the poor outcome of out-of-hospital resuscitation. Identification of PEA requires an ECG and some means to identify the absence of a pulse. No existing automatic external defibrillator (AED) (or implanted cardioverter defibrillator

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Pulseless electrical activity

[ICD]) is equipped with a pulse detector and unless the
rescuer views the ECG and palpates the pulse, PEA will be
missed and deterioration and death are likely. If the rescuer
identifies R waves in the ECG but no pulse is palpable,
prompt cardiopulmonary resuscitation (CPR) and advanced
life support are mandatory.

The amount of time that ventricular fibrillation (VF)
must last, without CPR support, to encounter PEA is, at
present, not well documented. The present animal study was
undertaken to identify the duration of unsupported VF
needed to encounter postdefibrillation PEA.

2. Methods and materials

All studies were performed on 9 anesthetized pigs ranging
in weight from 20 to 30 kg. Each animal was sedated,
intubated, and anesthetized with isoflurane and oxygen to a
depth of Guedel stage 3, plane 2. Femoral artery pressure and
lead II ECG were recorded. VF was induced electrically with
a right ventricular catheter electrode to which 2-millisecond
pulses at 50 per second with an intensity of 10 V were
applied. VF was confirmed by the appearance of fibrillation
waves in the ECG and a loss of pulsatile blood pressure. As
soon as VF was confirmed, the anesthesia was discontinued
and restored after ventricular defibrillation. VF was allowed
to persist for increasing 15-second increments (eg, 15, 30,
45, 60, etc). At the end of each increment, the ventricles were
defibrillated with transchest electrodes. If no R waves
occurred because of S-A node arrest or AV block, the
ventricles were paced with the right ventricular catheter
electrode used to induce VF. In this way, we could test for
PEA in all animals. Cardiopulmonary resuscitation was
applied with the chest thumper (Michigan Instruments Inc)
to recover the animals. Additional trials to identify PEA were
not attempted until the animal’s blood pressure returned to
the prefibrillation pressure. All data were entered into an
online computer (LABVIEW), whereas recordings were
made on a stripchart recorder.

Control blood gases and chemistries (pH, K+, Na+HCO3−,
Cl−, hematocrit, hemoglobin, SaO2 levels) were obtained
before the induction of VF and immediately after defibril-
lation using the chest thumper to provide circulation during
PEA. During these episodes blood gases and chemistries
were obtained.

VF wave frequency was measured on a second-by-
second basis on the animals carried to 180 seconds. This
was done to determine if VF wave frequency could be used
as a predictor of postdefibrillation PEA.

3. Results

Fig. 1 is a record of the ECG and blood pressure that
illustrates postdefibrillation PEA. In this record, there is total
AV block with an idioventricular rhythm, and the R waves
are not followed by blood pressure pulses (left and right). In
the center of the record, rhythmic chest compressions were
applied between START and STOP; note the blood pressure
pulses. On cessation of chest compression, PEA returned.

Fig. 2A is a histogram showing the number of PEA
occurrences versus the duration of VF using class intervals
of 25 seconds. Fig. 2B shows the same data expressed as the
percentage of PEA occurrences versus the duration of VF:
this figure was made by summing the data in the histogram.
shown in Fig. 2A. Note that by 80 seconds, there was an 18% incidence of PEA. By 180 seconds, the incidence of PEA was 100%.

Before the induction of VF, the blood gases and chemistries were all normal. In those animals in which PEA was present after defibrillation, the chest thumper was applied, and atrial blood samples were drawn immediately. Because the animal was mechanically ventilated with oxygen, the \( P_{O_2} \) and \( S_aO_2 \) were high. With a mean arterial pressure of 20 to 25 mm Hg provided by the thumper, the pH initially increased because of hyperventilation, then decreased because of supervening metabolic acidosis. The most striking feature was a continued rise in \( K^+ \) from a normal value of 4 mEq/L to 8 to 12 mEq/L, the magnitude of the increase being dependent on the duration of VF.

The frequency of the VF waves was measured, and shown in Fig. 3 is a typical example, showing the variation in frequency and a gradual decrease in mean frequency with the passage of time. Typically, the largest decrease in VF wave frequency occurred in the first minute. Fig. 4 summarizes the mean VF wave frequency versus the duration of VF out to 180 seconds. We were able to recover cardiac pumping in 1 pig with PEA when VF lasted for 180 seconds. Fig. 4 also shows the percent of postdefibrillation PEA versus the duration of VF using the data from Fig. 2B. By 60 seconds (the threshold for PEA), the mean VF wave frequency was 65% of the initial frequency.

4. Discussion

Decoupling of excitation and contraction in human hearts appears to have been reported first by Dorra [1,2] and Dorra et al [3] who described it in the atria after cardiversion with 250- to 300-J shocks. He called the phenomenon “dissociation electromechaniques,” that is, electromechanical dissociation. The modern term is PEA, and it can occur in the atria and ventricles; however, PEA is usually described in association with failure of the ventricles to contract after an R wave in the ECG.

In the present study, it was a surprise to encounter 100% PEA incidence after 180 seconds of untreated VF (no CPR). It is of interest to note in Fig. 3 the manner in which the VF wave frequency decreased with time. The threshold duration
of VF for PEA was 60 seconds. However, there is no sharp change in VF frequency at this time. The mean frequency at the onset of VF ($f_0$) was $10.1 \pm 2.1$ per second; at 180 seconds ($f_{180}$), it was $4.0 \pm 0$ per second. The mean ratio ($f_{180}/f_0$) is $0.35 \pm 0.02$. In all cases, the VF wave frequency decreased with the passage of time. The wide range of initial frequencies ($f_0$) and the manner of decrease in each animal make it impossible to use VF wave frequency as an indicator of how long VF was present. The progressive decrease in frequency is an indicator of the increasing difficulty of the myocardium to sustain VF caused by hypoxia. However, an increase in VF frequency during CPR may well indicate the effectiveness of myocardial oxygenation.

In a 29-dog study, Vincent et al [4] induced VF and allowed it to persist from 30 to 180 seconds, then defibrillated to identify the time when postdefibrillation PEA appeared. They found that after 120 seconds of VF, postdefibrillation PEA could be produced reliably. In the present pig study, it required 180 seconds of untreated VF to encounter postdefibrillation PEA in 100% of the trials. However, in a few instances, it was possible to produce PEA after 75 to 100 seconds of fibrillation.

The dominant frequency component in a power-frequency spectrum analysis of VF waves from 41 patients was reported by Stewart et al [5]. Data obtained in short-time segments showed that the dominant frequency decreased with the passage of time. At 3 seconds, the dominant frequency was 5.8 per second, and at 20 seconds, it was 2 per second. A plot of the frequency versus time data resembled a decaying exponential curve.

Power-spectra studies of the ECGs of dogs during VF were reported by Martin et al, [6] the objective being to identify any changes with the passage of time during VF. In the first few seconds of VF, the spectrum exhibited a narrow peak between 15 and 18 per second. In the following 40 seconds, the frequency increased, after which the frequency became low and irregular, losing its characteristics after 60 seconds. Except for the slight rise in frequency, the change is in general agreement with the instantaneous frequency data obtained in the present study.

In another study by the same authors to study electromechanical dissociation (PEA) using dogs, defibrillation was performed after successive periods of VF. In all dogs, PEA was observed after 90 seconds of VF. In the present swine study, 60 seconds was about the threshold for encountering PEA.

Using the Fast-Fourier Transform to analyze the ECG during VF in anesthetized dogs, Carlisle et al [7] reported that the dominant VF frequency with body-surface electrodes, recording from nonischemic hearts, was initially 9.9 ± 0.7 Hz, remained above 9 Hz for 70 seconds and then rapidly fell to 5 Hz.

The same authors measured the dominant frequency of VF waves, induced by acute coronary occlusion, to be initially $12.3 \pm 0.2$ Hz. The initial frequency is in agreement with the pig data in the present study.

A consistent finding in the present study was the association of PEA with a high K+ in the blood of all animals. That high potassium is a myocardial depressant has been known since 1883 when Ringer [9] showed that excess K+ arrested the heart in diastole. He also found that a high Ca++ arrested the heart in systole. In a series of carefully controlled experiments in which he varied the K+/Ca++ ratio, he found a combination that maintained cardiac function, thus was born Ringer solution.

In a study with human hearts, Singh et al [8] found that with 7 minutes of anoxia, K+ leaked out of myocardial cells.

The high values of K+ associated with prolonged untreated VF, we believe, are caused by the lack of tissue perfusion which impairs the ability of the metabolically driven membrane Na+/K+ ion pump. In the present study, postdefibrillation K+ rose as high as 8 to 12 mEq/L in animals in which VF was prolonged, indicating poor tissue perfusion because the SaO2 during postdefibrillation was 98% and the mean blood pressure was 25 mm Hg with CPR and PEA present. Clearly, poor tissue perfusion with oxygenated blood did not eliminate PEA in those animals that sustained VF for long periods. In other words, poor perfusion with oxygenated blood results in hyperkalemia.

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**References**


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