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EXPERIMENTAL PAPER

Post-resuscitation hemodynamics and relationship to the duration of ventricular fibrillation☆

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Summary
Objective: We have observed consistent hemodynamic patterns after restoration of spontaneous circulation (ROSC) after ventricular fibrillation (VF) cardiac arrest. We sought to characterize the time-course of these patterns, and to determine whether these differed based on duration of the VF insult.
Methods: We performed a retrospective review of data from a randomized animal experiment that was conducted in an AAALAC-approved animal laboratory. We used mixed-breed domestic swine of either sex. Animals were anesthetized and instrumented for continuous recording of ECG and blood pressures. VF was induced electrically and allowed to progress for various times ranging from brief (22 s) to moderate (less than 3 min) to prolonged (3—10 min). All animals were initially shocked (150 J) up to three times. If ROSC was not achieved on the three initial shocks, a standardized treatment protocol was followed. We defined cardiovascular collapse as a SBP < 90 mmHg sustained for 1 min. For statistical purposes, we classified animals as having VF of <3 min, or >3 min duration. Data were analyzed with Fisher's exact test and survival analysis.
Results: A hyperdynamic phase, consisting of very high-blood pressures and tachycardia, was seen in all animals immediately after ROSC. This lasts from 1 to 4 min. Post-resuscitation cardiovascular collapse occurred in 2/7 (29%) animals in the <3 min group and 13/14 in the >3 min group (93%) $p = 0.006$. Onset of cardiovascular collapse was highly related to duration of VF (log-rank $p = 0.004$).
Conclusions: There are two distinct phases of hemodynamic change after resuscitation of VF. The first phase is a brief hyperdynamic phase. The second phase is either stabilization or cardiovascular collapse. When VF is brief, blood pressures often return to normal without exogenous support. When VF was prolonged animals were rescued with exogenous pressor. Healthcare providers should be prepared to provide pressor support for patients having ROSC after prolonged VF.

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Introduction

Sudden cardiac death remains the most significant public health problem in the United States.[1] Approximately 325,000 such deaths occur annually, with only 6% of these patients surviving to hospital discharge.[2,3] Many patients who suffer an out-of-hospital cardiac arrest (OOHCA) have their pulses restored in the field but do not survive to be admitted to the hospital. For example, of 1474 OOHCA patients treated by the City of Pittsburgh EMS, 32% had pulses restored in the field while only 25% still had pulses at arrival to the emergency department (a 22% loss of pulses after initial resuscitation).[4]

This loss of circulation may be explained in part by post-resuscitation instability that often occurs after the prolonged global ischemia that occurs during cardiorespiratory arrest. This instability is the result, in part, of left ventricular dysfunction and loss of vascular tone.[5–7] In our laboratory, we have observed post-resuscitation hemodynamic patterns that seem to be dependent on the duration of the antecedent ventricular fibrillation (VF). Better understanding of these patterns might help healthcare providers anticipate the need for exogenous pressor support, thereby preventing the loss of pulses during the transport of OOHCA patients.

We sought to determine whether the nature and time-course of these post-restoration of spontaneous circulation (ROSC) hemodynamic patterns were temporally associated with the duration of VF. We also sought to describe the phenomenon of post-resuscitation cardiovascular collapse. We hypothesized that post-resuscitation cardiovascular collapse would be associated with the duration of VF, i.e. that brief periods of VF would have delayed or no post-resuscitation cardiovascular collapse, while prolonged VF would have rapid onset of post-resuscitation cardiovascular collapse.

Materials and methods

This investigation was approved by the University of Pittsburgh Institutional Animal Care and Use Committee (IACUC). We retrospectively analyzed the data from 24 mixed-breed domestic swine of either sex that were a subset of another experiment.[8] These animals were selected because they both represented variety in the duration of VF and were treated with immediate defibrillation attempts as the first therapy (i.e. these animals did not receive any drug treatment before the first 1—3 rescue shocks).

The animals ranged in mass from 19.5 to 25.7 kg. The preparation of these animals has been previously described in detail.[8] In brief, animals were sedated with ketamine and xylazine, anesthetized with alpha-chloralose, orotracheally intubated, and paralyzed with pancuronium. Micro-manometer-tipped catheters (Mikro-Tip Catheter Transducers SPR-471A and SPC-370-S, Millar Instruments, Houston, Texas) were advanced into the aorta and right atrium. Pressures were recorded continuously throughout the experiments using a computerized data acquisition system and software package (Chart, v.5.3, ADInstruments, Castle Hill, Australia). Arterial blood gases were drawn periodically to establish the physiologic stability of the preparation, and any time the ventilator was adjusted (i-STAT Portable Clinical Analyzer, Heska Corporation, Waukesha, WI).

At the end of the preparation, VF was induced with a 3 s, 60 Hz, 100 mA transthoracic shock. VF was then untreated until the animals' ECG displayed a predetermined level of organization (scaling exponent value). Animals were then treated with up to three fixed-dose countershocks (150 J, biphasic waveform), depending on the post-shock ECG rhythm. If these shocks failed, mechanical CPR was begun (Thumper, Model 1007, Michigan Instruments, Grand Rapids, MI) and standardized care was given.[8]

We defined ROSC as an organized post-shock ECG rhythm combined with a systolic blood pressure of at least 90 mmHg sustained for 1 min continuously. We defined the hyperdynamic phase, *a priori*, as post-ROSC systolic blood pressure of 150 mmHg or greater. We defined post-resuscitation cardiovascular collapse as a post-ROSC systolic blood pressure below 90 mmHg sustained continuously for at least 60 s. This was also the point at which exogenous pressor support with norepinephrine was begun. For the purposes of time-to-event analyses, we dichotomized the classification of VF duration into <3 min VF and >3 min VF.

We compared the proportions of animals attaining ROSC and the proportion of animals experiencing post-resuscitation cardiovascular collapse in each group and compared these with Fisher's exact test. We compared time to post-resuscitation cardiovascular collapse using survival analysis.[9]

Results

The distribution of VF duration for the two groups is shown in Figure 1. Twenty-one of 24 animals achieved ROSC; 7/7 (100%) in the <3 min VF group and 14/17 (71%) in the >3 min VF group. Time-to-event (stabilization or cardiovascular collapse) data were not available for three animals and these were excluded from further analyses.

There were two distinct phases of post-ROSC hemodynamics. First, there is an immediate hyperdynamic phase characterized by very high-aortic blood pressures combined with tachycardia. This was observed in all animals that had ROSC, regardless of duration of VF, and lasts for 1—4 min.

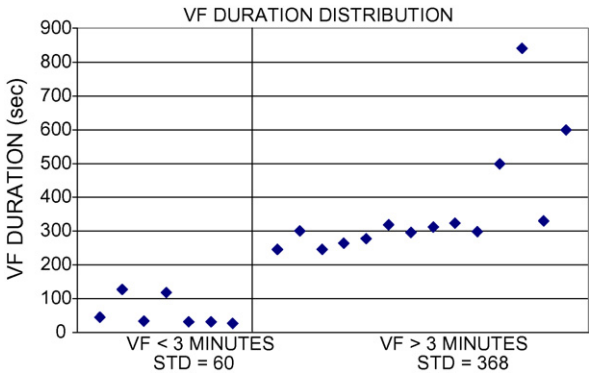

Figure 1 Distribution of the duration of VF, in seconds, for animals (STD, standard deviation in seconds for group) categorized as having either <3 min VF or >3 min VF.