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The Stability of Electrically Induced Ventricular Fibrillation

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Abstract:[1]

The first recorded heart rhythm for cardiac arrest patients can either be ventricular fibrillation (VF) which is treatable with a defibrillator, or asystole or pulseless electrical activity (PEA) which are not. The time course for the deterioration of VF to either asystole or PEA is not well understood. Knowing the time course of this deterioration may allow for improvements in emergency service delivery. In addition, this may improve the diagnosis of possible electrocutions from various electrical sources including utility power, electric fences, or electronic control devices (ECDs) such as a TASER® ECD.

We induced VF in 6 ventilated swine by electrically maintaining rapid cardiac capture, with resulting hypotension, for 90 seconds. No circulatory assistance was provided. They were then monitored for 40 minutes via an electrode in the right ventricle. Only 2 swine remained in VF; 3 progressed to asystole; 1 progressed to PEA. These results were used in a logistic regression model. The results are then compared to published animal and human data.

The median time for the deterioration of electrically induced VF in the swine was 35 minutes. At 24 minutes VF was still maintained in all of the animals. We conclude that electrically induced VF is long-lived — even in the absence of chest compressions.

INTRODUCTION:

The incidence of ventricular fibrillation (VF), as the presenting rhythm of field cardiac arrests, is decreasing while the incidence of asystole is increasing.[1] While VF is typically treatable electrically, asystole and PEA are not. The return to normal neurological function among patients presenting as PEA or asystole is a dismal 0.4%.[2] Studying the deterioration of VF with humans is confounded by the fact that about 30% of cardiac arrests — occurring among patients who are being monitored for chest pains — are asystolic.[3, 4] These continuously monitored patients show no evidence of pre-asystolic ventricular tachycardia (VT) or VF.

The mechanism of a cardiac arrest beginning as asystole is not well understood.[5] However, there is a reasonable mechanism for VF deteriorating to asystole. VF, regardless of the precipitating factors, leads to myocardial ischemia. (Of course, an acute ischemic event may have been the trigger to begin with.) Lactic acid increases due to the anaerobic glycolysis and $C0_2$ increases 4x within 30 minutes.[6] The myocardial pH shifts by more than 0.5.[7] When the extracellular myocardial pH reaches 6.5, the depolarization potential is essentially gone (< 10 mV).[8] As this point (or earlier) asystole ensues.

Understanding the time course of this deterioration may allow for improvements in emergency service delivery. In addition, this may improve the diagnosis of possible electrocution.

METHODS and RESULTS

The protocol was approved by the IACUC for the University of Alabama at Birmingham. Swine (n=6) weighing 20-25 kg were initially anesthetized with telazol/xylazine (4.4 mg/kg) and intubated. An arterial line was inserted for pressure measurements and an electrogram catheter was inserted into the right ventricle. The animals were anesthetized with isoflurane (1-3% inhalation). Arterial blood gases and electrolytes were measured every 30 – 60 minutes.

An adjustable probe assembly (Figure 1) was placed in an intercostal space directly over the right ventricle. The probe was advanced under fluoroscopy until the tip was 10 mm from the epicardium. A remote return electrode was placed in the lower abdomen.

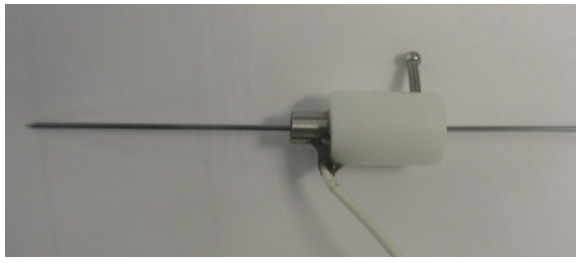

Figure 1. Adjustable depth current delivery probe.

A custom waveform generator was used that can generate rectangular pulses of varying durations, pulse rates, and charges or voltages. The pulse duration was fixed at

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100 µs as that is a common pulse width for both the electronic control device (ECD) and the electric fence.[9, 10] This was sufficient to achieve rapid hypotensive capture via epicardial stimulation thru the adjustable probe. The pulse rate was set at 20 Pulses Per Second (PPS). This is consistent with the 15-25 PPS rates used in existing.[10, 11] The stimulation to the epicardium was maintained until VF was induced. This typically required at least 90 seconds.[12]

The transition times and resulting rhythms are shown in Table 1.

Table 1. Results for the 6 swine.

Subject	Transition Time (minutes)	Resulting Rhythm
1	29.08	Asystole
2	24.33	Asystole
3	40	Still in VF
4	40	Still in VF
5	24.43	Asystole
6	38.83	PEA

These data were fitted with a logistic regression model. Besides the transition times shown in Table 1, each subject had a VF datum entered for 0, 10, and 20 minutes. The results are shown in Figure 2. The data were well fit by the regression model. (Wald's U = .26, p = .01). The model-interpolated median deterioration time was 35 minutes. This was consistent with the raw median from the data of 34 minutes.

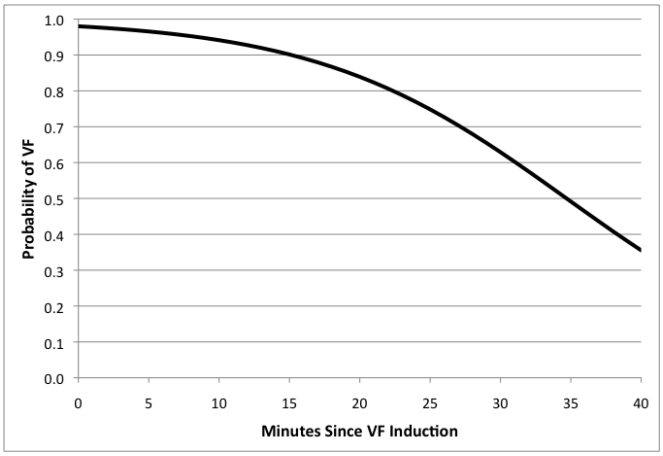

Figure 2. Logistic regression fit to the swine data for electrically-induced VF.

Comparison to Human Cardiac Arrest Data (Non-Electrically Induced)

Obviously, there are no similar data available for humans. Hence we compared our results to epidemiological studies of human cardiac arrest rhythm changes.

Waalewijn et al studied the presenting rhythm of 873 patients who experienced a witnessed cardiac arrest.[13] The breakdown by rhythm and bystander CPR is given in Table 2. They fit the data to a logistic regression model using the time-to-recording and CPR as predictors.

Table 2. Presenting rhythms in Waalewijn study.

	VF	Asystole	Other	Total
Bystander CPR	313 (66%)	70 (15%)	93 (19%)	55%
No CPR	200 (50%	79 (20%)	118 (30%)	45%
Total	513 (59%)	149 (18%)	211 (24%	

Their model predicted that the initial rhythm (at the time of the arrest) was VF in 73% of the cases and asystole in 6%. We normalized their VF vs. time model, by dividing the VF percentage values by 0.73 (since only 73% of patients began in VF), to arrive at the model for the maintenance of non-electrically induced VF as shown in Figure 3. For any given level of confidence of VF maintenance, it is seen that the number of minutes was at least doubled (2x) by the use of CPR.

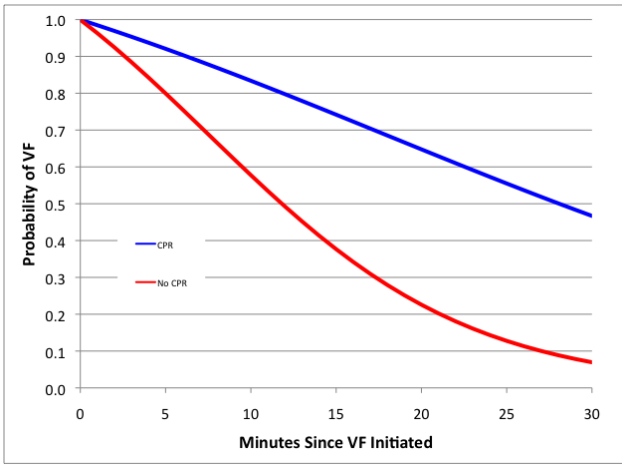

Figure 3. The probability of the maintenance of VF after non-electrically induced VF cardiac arrest in Waalewijn.

Holmberg et al studied 1100 heart-disease patients with witnessed cardiac arrests that received CPR.[14] They gave a linear fit for the probability of VF vs. time with a initial rate of 85% and a degradation of 1.06% per minute. (correlation coefficient not given)

Hallstrom et al studied 515 witnessed out-of-hospital cardiac arrests.[15] Of patients with recorded presenting rhythms, they were able to estimate the time to the tracing in 93% of the cases. CPR was provided to 27% of the patients and the data were not broken down by the absence or presence of CPR. We fit the probability of VF by a mono-exponential of the time to tracing. ($r^2 = .75$)

We normalized the VF rates of the Holmberg and Hallstrom studies by their initial rhythm predictions to obtain estimates of the rate of degradation from an initial VF rhythm. This is shown in Figure 4 along with the

normalized Wallewijn values. Note that the median time for the disappearance of VF is > 30 minutes.

However, the disappearance of non-electrically induced VF does not necessarily mean that the VF deteriorated to asystole as it may have changed to, say, PEA. Thus we used Waalewijn's model to predict the probability of *new onset* asystole by subtracting 6% from his model for the presence of asystole. Hallstrom reported on the asystole rate as well — which data were best fit by a parabolic curve. Holmberg did not give asystole rates. These asystole results are shown in Figure 5. These data show that the median time for non-electrically induced VF to deteriorate to asystole is also > 30 minutes. Extrapolation suggests that the median time is approximately 32 minutes.

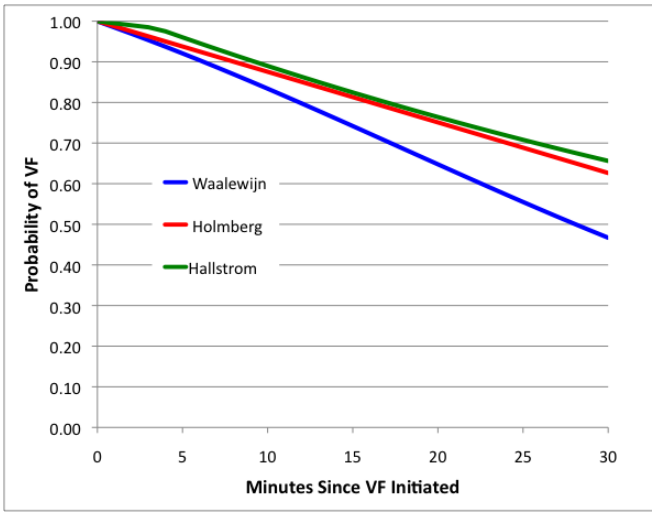

Figure 4. The probability of the maintenance of non-electrically induced VF after a VF cardiac arrest.

Based on the Waalewijn data, we can estimate that, with CPR, about 10% of humans in *ischemically-induced* VF will deteriorate to asystole in 12 minutes.

DISCUSSION:

We believe that this is the first report of the time for VF maintenance after electrical induction with prolonged hypotensive capture. Consistent with most other reports we found no deterioration to asystole or PEA in 20 minutes of electrically-induced VF.[16, 17] Wiggers (1930) found a mean VF duration of 24 minutes in canines.[18]

We have previously reported that an analysis of Holter recordings showed a median time, for non-electrically induced VF to deteriorate to asystole, of 19 minutes in humans.[19] Only 1 of those cases (42 minutes to asystole) was electrically induced. To the best of our knowledge, none of those cases received CPR. Our analysis of epidemiological observational data suggests that the median time for the deterioration of VF to asystole is just over 30 minutes. Note that 83% of these patients received CPR.

A confounding effect is the conversion of asystole into VF by CPR. The rate of such a conversion has variously been reported between 10-20%.[20, 21] While this is typically reported with concomitant use of atropine and sympathetic agents, it is not clear that such pharmacological therapy plays a major role. Hence, we must consider the possibility of some of the late "presenting" VF was actually asystole converted to VF during CPR. Assuming that 15% of the initial cardiac arrests were due to asystole, and assuming a 15% conversion rate of asystole to VF, then about 2% of the late VF may have been due to converted asystole. This would reduce our estimate of the median time to asystole by about 1 minute, resulting in a median time to asystole of 31 minutes.

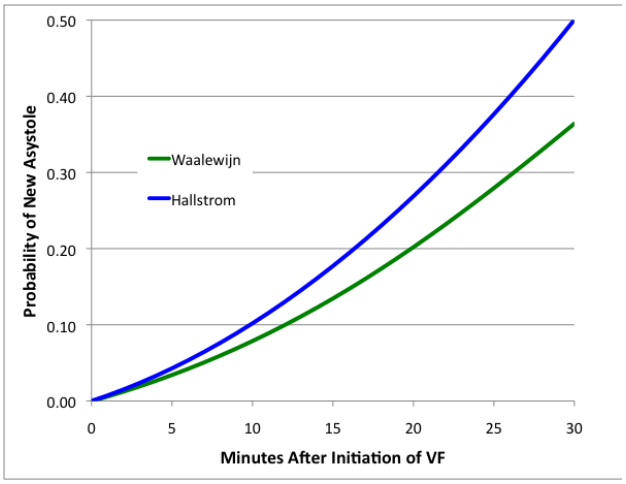

Figure 5. Rate of new asystole as a function of the duration since a VF cardiac arrest.

The differences between the animal and human data are summarized in Table 3. Despite differences in size, species, premorbid health status of the heart and circumstances, the median times to asystole (or PEA) are remarkably close. The humans had the disadvantage of cardiac arrests that in many cases were presumably induced by myocardial ischemia. A significant proportion of the others likely occurred on the basis of hypertensive heart disease. Thus, many of the cardiac arrests in humans occur in hearts having pathologically hypertrophic left ventricles. Both of these circumstances would tend to cause earlier myocardial acidosis and thus may tend to hasten deterioration to asystole. Conversely, most of the humans received circulatory assistance via CPR while the animals had none.

Table 3. Comparison of Animal and Human Studies

	Induction	Ventila-tion	Circula-tory Support	Elec-trodes	Medi-an Time in VF
Ani-mal	Electrical	Ventila-tor	None	Internal	35
Hu-man	Primarily ischemic	CPR in 83%	CPR in 83%	External	31

How do we estimate the time for deterioration of electrically-induced VF to asystole when chest-compressions are performed? Certainly, this should be longer than the times for animals without compressions (15 minutes to 10% asystole rate). We have shown elsewhere that the use of chest compressions extends the time for successful defibrillation by 40%.[22] Note that Wallewijn found that CPR doubled (2x) the time for VF to deteriorate in humans. Since chest compressions delay the metabolic deterioration of VF by about 40% (in swine) and 100% (in humans) we can estimate that the time for a 10% rate of VF conversion to asystole at about 21-30 minutes.

With the same reasoning we estimate that the median time for electrically-induced VF to deteriorate to asystole to be 49-70 minutes.

There are few cases of more rapid VF to asystole deterioration have been reported. An 18-minute case was reported by Panidis.[23] A 16-minute deterioration was reported by Twidale (5 minutes of polymorphic ventricular tachycardia and 11 minutes of VF).[24] Neither of these patients received CPR.

Conversely, there are some dramatic cases of patients being kept in VF for long durations with compressions. A 54 year-old man received CPR for 96 minutes until resuscitation and was discharged without neurological deficit.[25] The record may be 110 minutes (with a successful outcome) but that involved open-chest massage.[26]

CONCLUSIONS:

We have studied the time for electrically-induced ventricular fibrillation to deteriorate into asystole or PEA in ventilated animals. The median time was 35 minutes. No animals deteriorated in less than 24 minutes.

Although occasional instances, in humans — of more rapid VF to asystole deterioration — have been noted, the median time for deterioration to asystole or PEA is estimated at 31 minutes. However, the point at which 90% of the cases have not degraded to asystole is approximately 12 minutes. The shorter duration is most likely due to the myocardial ischemic acidosis developing before the cardiac arrest.

Based on the existing data, we estimate that it requires about 21-30 minutes for electrically-induced VF to deteriorate to asystole with a 10% probability assuming some chest compressions. We estimate the median time to be 49-70 minutes.

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