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Peri-shock Pause Intervals and Rearrest after Out-of-Hospital Cardiac Arrest

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Abstract

Background: The loss of pulses after successful return of spontaneous circulation (ROSC) in out-of-hospital cardiac arrest (OHCA) is known as rearrest (RA). The causes of RA are not well understood.

Objectives: To investigate the association between shock pause intervals and RA.

Methods: Data from treated OHCA with ROSC and one or more defibrillation attempts were obtained from one site of the Resuscitation Outcomes Consortium. All analyses were conducted internally. Data available for analysis included cases spanning 2006-2008 and 2010-2011. Defibrillator tracings were used to calculate both components of the peri-shock pause (PSP) interval: the pre- (preSP) and the post-shock pauses (postSP). RA and no-RA shock pauses were compared and independent associations between shock pause intervals, patient characteristics and RA were assessed with the appropriate statistical tests.

Results: Analysis included 241 shocks from 101 cases. Forty-one cases (41%) had RA. RA vs. no-RA median (IQR) shock pauses in seconds were: preSP 13.5 (6.0-18.0) vs. 15.0 (10.9-21.5) ($p = 0.121$); postSP 6.0 (3.5-8.2) vs. 8.7 (4.5-13.9) ($p = 0.053$); and PSP 18.0 (12.3-24.0) vs. 24.0 (16.7-30.2) ($p = 0.022$). Considering all possible shock pause durations, shock pause lengths and various patient characteristics were not associated with RA. If 30 seconds or shorter, the preSP (OR: 0.90 {0.82-0.98}) and postSP (OR: 0.89 {0.79-0.99}) were related to RA.

Conclusion: Shock pause length was inversely associated with RA when shock pause intervals were limited to 30 seconds or less. Shock pauses and RA were not associated when all durations of shock pauses were considered.

Keywords

cardiac arrest; cardiopulmonary resuscitation; emergency medical services; shock pause interval; prehospital intervention

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Introduction

Rearrest (RA) occurs when a patient loses pulses following successful attainment of return of spontaneous circulation (ROSC). Previous studies have investigated the rate of occurrence of RA in out-of-hospital cardiac arrest (OHCA), as well as its relationship to first identified electrocardiogram (ECG) rhythm. Reported rate of RA ranges from 36 to 79%.¹⁻⁵ Despite this relatively high incidence, the precise mechanisms and contributing factors responsible for RA remain undetermined. While there is physiological plausibility for the role of pre-existing pathology in RA, there is no direct evidence in the literature to support or refute this assumption. Similarly, there is little evidence for or against the role of emergency medical services (EMS) personnel behaviors affecting the occurrence of RA. However, critical interventions delivered by EMS, including cardiopulmonary resuscitation (CPR), defibrillation, and airway management, before and after ROSC have been shown to affect patient outcomes.⁶⁻⁷ Moreover, unlike patient pathology, paramedic behaviors can be altered systematically.

The limited body of literature on this topic compelled us to explore possible EMS procedural contributors to RA, beginning with procedural no-flow time, that is, gaps in delivery of chest compressions during resuscitation due to delivery of other treatments. A common source of procedural no-flow time is the peri-shock pause (PSP), which occurs when CPR is interrupted for the delivery of defibrillation.⁸ The interval is calculated as the elapsed time from the last pre-shock compression to the first post-shock compression for any given defibrillation shock. Increasing duration of the PSP interval has been shown to adversely affect survival to hospital admission and survival to hospital discharge.⁹⁻¹¹ However, no studies that we know of have investigated the association between shock pause intervals and RA.

The goal of this study was to determine if shock pause intervals were associated with RA. We hypothesized that increasing shock pause length would be associated with RA, owing to the acute effects of no-flow time on the heart.

Materials and Methods

We obtained approval from the Institutional Review Board commencing this study. Case data were retrospectively obtained from the Pittsburgh site of the Resuscitation Outcomes Consortium (ROC), a multicenter clinical research consortium that has ten sites across North America and investigates cardiac arrest and severe traumatic injury. Qualifying cases were non-traumatic, EMS-treated OHCA that occurred in the Pittsburgh, Pennsylvania area from October 2006 to December 2008 and January 2010 to December 2011 and contained at least one instance of prehospital ROSC. We did not exclude pediatric cases. Data collected during the ROC PRIMED clinical trial in 2009 were excluded. For all cases, multi-modal defibrillator downloads, including ECG, compression depth, transthoracic impedance, and continuous audio recording were the primary data sources.

ROSC, RA, and shock pause intervals were ascertained through analysis of the electronic defibrillator downloads with custom software written in Matlab 2011b (MathWorks, Natick,

MA). For this study, ROSC was defined as the resumption of pulsatile mechanical and electrical function of the heart that lasted at least 60 seconds. The Utstein criteria list 30 seconds as any instance of ROSC and 20 minutes as “sustained ROSC.”¹² Using 20 minutes would severely limit the number of cases available for this study, as many of our out-of-hospital resuscitations lasted less than an hour and several of our data sources were only available up to the first 20 minutes of resuscitation. Instead we set 60 seconds as our ROSC criterion for ease of data abstraction and because it encompassed Utstein’s criterion for “any ROSC.” We identified ROSC when the ECG trace presented an organized rhythm, coupled with paramedics verbalizing confirmation of pulses on the defibrillator audio recording, when available, or the absence of chest compressions and rescue shocks. RA was identified when the patient presented a lethal dysrhythmia (e.g., ventricular fibrillation) in the ECG trace after obtaining ROSC, and only RA in the prehospital setting was studied; we did not investigate in-hospital RA. Using the compression depth trace, transthoracic impedance trace, and defibrillator-generated shock-delivery annotation, we were also able to identify RA when paramedics resumed CPR or delivered a rescue shock following ROSC. Lastly, RA could be further confirmed if paramedics verbalized “loss of pulses” on the defibrillator audio recording. All ROSC and RA event identifications were confirmed independently by two separate investigators (ACK / DDS).

We identified all shocks by defibrillator shock annotation and calculated the shock pause intervals to the nearest second as follows: the pre-shock pause (preSP) was determined to be the interval from last pre-shock compression to shock delivery, and the post-shock pause (postSP) was the interval from shock delivery to next chest compression. The PSP was then the sum of the two intervals; that is, it was the interval from last chest compression through shock delivery to the next post-shock compression. In the event that a patient had ROSC following a rescue shock, there was technically no postSP, and the preSP and PSP were equal in length. Case averages for each of the three shock pause intervals were calculated and used for statistical analyses. We excluded cases that had stacked shocks – that is shocks that were delivered rapidly, in sequence without intervening chest compressions – as they currently and during the time period over which study data spans, not part of standard Advanced Cardiac Life Support guidelines. We excluded individual shocks and their associated shock pause intervals when the shocks were not preceded by chest compressions as the physiologic conditions in such cases may be mechanistically distinct from those involving pre-shock compressions. Finally, we excluded shocks that occurred after first ROSC as they may be physiologically different than those preceding ROSC.

Chest compression fraction (CCF) is the proportion of time in which chest compressions are performed during each minute of on-going resuscitation and can be used as a metric of CPR quality and as a predictor of survival.¹¹ For each case, average CCF was available for up to the first 20 minutes of resuscitation from minute-by-minute CCF measures collected as part of the standard ROC data abstraction mechanism. Periods without CPR did not contribute to case average CCF.

For statistical analyses, we used Microsoft Excel (Microsoft, Redmond, WA) and Stata 12 (Stata Corp, College Station, TX) and uniformly used an alpha level of 0.05 as the criterion for statistical significance. Two-tailed t-tests and chi-squared tests were used where

appropriate to compare means and proportions of patient characteristics. The distributions of shock pause intervals were compared between RA and no-RA groups with the Kruskal-Wallis test due to non-normality. Multiple logistic regression models were used to assess the association between case average shock pause intervals and RA while controlling for key procedural and condition variables, including age, sex, EMS witness status, pre-/post-2010 International Liaison Committee on Resuscitation Guidelines status, and shockable first EMS-assessed cardiac arrest rhythm (coded as shockable/non-shockable). PostSP was not always present due to a lack of compressions after some successful shocks. Therefore, the multiple logistic analyses would only include those cases with an available case average postSP. While we did not in practice exclude shock pauses from this study due to extremity of duration, we had some interest in delineating what might be “normal” shock pause duration from those that might be unusually long due to indeterminate procedural complications. Therefore we constructed two separate logistic regression models incorporating the covariates described above. Model 1 included all shock pause intervals observed. Model 2 excluded preSPs and postSPs of greater than 30 seconds. We acknowledge that this particular set of constraints is arbitrary, however we believe as an arbitrary choice it presents a reasonable limitation that fits the procedural timing of a field resuscitation, which is constructed in cycles of arbitrary length.

Results

One hundred and thirty-nine cases containing ROSC were identified as having received at least 1 EMS-delivered rescue shock at any point during the course of resuscitation. Thirty-eight cases were excluded due to stacked shocks, absence of shocks prior to first ROSC, or uniform absence of preshock chest compressions. In the remaining 101 cases, a total of 241 shocks with preceding chest compressions and occurring prior to first ROSC were available, including 241 preSPs/PSPs and 232 postSPs. See Figure 1 for our consort diagram.

The mean (SD) age of our sample was 65.1 (14.9) years, with ages ranging from 27 to 94 years, and 64 subjects (63%) were male. See Table 1 for selected case characteristics stratified by RA status. One case (1%) occurred in 2006, 18 (18%) in 2007, 15 (15%) in 2008, 31 (31%) in 2010 and 36 (36%) in 2011. See Table 2 for shock pauses and outcomes stratified by year.

RA of any presenting ECG rhythm occurred in 41 cases (41%), with an average (range) of 1.4 (1-3) RAs per person in the RA subset. The average time from first ROSC to first RA was approximately 6.5 minutes. Of the 41 RA cases, 28 (68%) arrived at the ED without a pulse after achieving ROSC. The overall survival rate was 29% ($n=29$). For the 41 cases with RA, there was a 24% ($n=10$) survival rate, while the no-RA group had a survival rate of 32% ($n=19$). These rates were not significantly different ($p=0.427$).

When all cases were investigated, the mean (SD) CCF was 67% (14%). When stratified by RA status, the mean (SD) CCF for the RA group was 67% (14%) while the non-RA group's mean (SD) CCF was 66% (12.6) ($p=0.713$). Thirty-seven cases (37%) had a non-shockable first rhythm. Of those, 22% ($n=8$) had pulseless electrical activity, 54% ($n=20$) had asystole, and 24% ($n=9$) had a non-shockable first rhythm as determined by an automated

external defibrillator. The RA group had a significantly higher median (IQR) number of shocks per case than the no-RA group (4 (2-6) vs. 2 (1-3.5, $p = 0.001$).

Overall median (IQR) shock pause intervals were 14 (9-21) for preSP, 7 (4-12) for postSP, and 20 (16-27) for PSP. Multiple logistic regression results are shown in Table 3. In Model 1, which included all observed shock pause intervals, neither preSP nor postSP were significantly associated with RA. However, in Model 2, which was constrained to only preSPs and postSPs less than or equal to 30s, both preSP (OR: 0.90, 95%CI: 0.82 – 0.98) and postSP (OR: 0.89, 95%CI: 0.79 – 0.99) were inversely associated with RA. In the same models, no other covariates were associated with RA.

Discussion

Rearrest may conceivably stem from multiple causes with varying contributions that are not necessarily corrected by resuscitation. In this study, we hypothesized that the procedural no-flow time associated with defibrillation would be associated with patients' likelihood of experiencing an RA. While it has been established that increasing no-flow time diminishes the likelihood of long-term survival after OHCA and increases the likelihood of irreversible brain damage in survivors, it was unclear whether the same mechanisms underlying those phenomena would also contribute to RA.^{13–14} Our results show that PSP duration was associated with incidence of RA, however quite unexpectedly the direction of this association was inverse, indicating that RA was less likely as PSP intervals increased. Critically, in this same sample we found no association between RA and survival, greatly limiting the consequences of the observed association between PSP and RA.

Within the paradigm holding that increasing no-flow time is generally deleterious to the patient, there is no obvious physiologic mechanism underlying the finding that shorter preSPs and postSPs would both increase the probability of RA. One could speculate about the postSP independently, which is temporally proximal to ROSC when a rescue shock is successful, occurring at the junction between chest compressions and spontaneous cardiac contraction. Overlap between chest compressions and a spontaneously beating heart may occur during this phase until ROSC is identified by the provider. In this scenario, duration of the postSP dictates roughly how long the heart has been beating when the post-shock chest compressions resume. The postSP then becomes a parameter for what might be a mechanism for induction of RA by compression of a beating heart. Tempting though this hypothetical mechanism may be, there is little supporting evidence for it in our data or the literature. It is more probable that shorter PSP intervals correlate with some unmeasured treatment-related factor that was not accounted for in our study, such as drug administration – including details of agent, dosage and timing –, airway management, or even paramedic unit experience or proficiency.

The dearth of literature on the topic of prehospital, all-rhythm RA as well as the lack of intersection between this literature and the growing body of studies investigating the relationship between PSP intervals and patient outcomes provide us with little true context in which to evaluate and compare our findings. From other PSP studies, we can really draw only one albeit imperfect comparison. That is, the PSP intervals that we observed in our

study were comparable in magnitude to those observed in other studies. Published estimates of median prehospital PSP intervals range from 3 to 16 seconds for the preSP and 4 to 38 seconds for the postSP.^{7, 15–16} This can only serve to give us assurance that the observed PSP intervals in our study were not wildly different from those observed elsewhere.

Moreover, from the particular cohort considered in our study, i.e. cases that all had prehospital ROSC, we cannot draw comparisons to other studies' findings regarding the relationship between PSP intervals and outcomes such as survival to hospital discharge or neurologic function. It is because of our cohort's unique ROSC requirement that normally significant predictors such as shockable first rhythm and witnessed status are not associated. We excluded cases that lacked any prehospital ROSC, and thereby may have removed some of the benefits that stem from having a shockable first rhythm or a witnessed arrest. The cases that remained in our cohort had nearly identical rates of shockable first rhythm between RA and no-RA groups, and similarly, the rate of witnessed status was very low for both groups. CCF was suboptimal for our cohort overall, but we are unable to directly associate certain pauses in CPR with specific procedures or other interruptions. Further, CCF did not differ significantly between the two groups, which may have deranged the associations we found between PSP, RA and survival.

Limitations

Our study had several limitations. Most importantly, we only examined the procedural no-flow time surrounding shock delivery. We did not investigate other sources of no-flow time, such as pulse checks, ventilation breaks, or moving the patient. In theory, no-flow time should be reflected in CCF, for which we controlled in our analyses. However, the validity of this surrogate may be mitigated by the process of averaging chest compression fraction across an entire case, which may mask critical periods of downtime. Calculation of absolute no-flow time could better approximate the true dose of "extra" ischemia imparted to the patient by provider CPR pauses. Future studies should consider all sources of no-flow time and the relationship of this more complete metric to RA. Additionally, for the purpose of this study, we analyzed case average shock pause intervals. Therefore our analyses reflect not the variance between individual shocks, but the variance of case average shock pause intervals between cases. This may have biased our conclusions toward the null. Our study also did not examine specific prehospital interventions or downstream in-hospital interventions, and data regarding post-OHCA interventions were extremely limited for this cohort. Additionally, our dataset exhibited a general lack of diversity in shock pauses across both the RA and no-RA groups. Pauses surrounding defibrillating shocks, or perhaps overall ischemic downtime, may have to be much longer than those we encountered in our dataset to show a definitive effect on RA. We cannot account for existing patient comorbidities or varying in-hospital treatments and their effects on survival to hospital discharge. Finally, our small number of cases came from a limited geographic area and temporal period, which may limit generalization to other populations.

Conclusions

In this limited study, increased shock pause length was associated with decreased probability of RA, but only when shock pause intervals were limited to 30 seconds or less. Shock pauses and RA were not associated when all durations of shock pauses were considered. The association between shock pause intervals and RA in this study was counterintuitive, and more research is necessary to elucidate the relationship between the two factors in OHCA.

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Article Summary

1. **Why is this topic important?** Every year, over 350,000 people in the US alone suffer from an out-of-hospital cardiac arrest (OHCA). Survival from OHCA is very low, and is estimated to be less than 10%. New methods and insights are needed to improve this dismal survival rate. Both shock pauses and rearrest (RA) have been shown to be correlated with poor outcomes.
2. **What does this study attempt to show?** This study attempts to elucidate the relationship between shock pause length and rearrest, two factors that are associated with poor OHCA outcomes.
3. **What are the key findings?** The main finding of this study is that some shock pauses are associated with an increased likelihood of RA.
4. **How is patient care impacted?** As indicated by other studies, shock pause length should be minimized, and healthcare providers should be vigilant of length pauses, which could be detrimental to OHCA patients.

Figure 1.
Consort Diagram

Table 1.

Case Characteristics Stratified by RA Status

Characteristic	All	RA	No-RA	p-value
	(n=101)	(n=41)	(n=60)	
<i>Age, y, mean (SD)</i>	65.1 (14.9)	66.0 (13.5)	64.5 (15.8)	0.625
<i>Male, % (95% CI)</i>	63.4 (53.8-72.9)	68.3 (54.0-82.5)	60.0 (47.6-72.4)	0.396
<i>EMS Witnessed, % (95% CI)</i>	8.0 (2.6-13.4)	5.0 (0.0-12.1)	10.0 (2.2-17.8)	0.367
<i>First ECG Rhythm VF/VT, % (95% CI)</i>	63.4 (53.8-72.9)	63.4 (48.0-78.8)	63.3 (50.8-75.9)	0.993
<i>Number of Shocks, median (IQR)</i>	3.0 (1.0-5.0)	4.0 (2.0-6.0)	2.0 (1.0-3.5)	0.001
<i>Survival to Hospital Discharge, % (95% CI)</i>	28.7 (19.7-37.7)	24.4 (10.7-38.1)	31.7 (19.5-43.8)	0.427
<i>Post-2010, % (95% CI)</i>	66.3 (57.0-75.7)	61.0 (46.0-75.9)	70.0 (58.4-81.6)	0.346
<i>Chest Compression Fraction, mean (SD)</i>	66.6% (64.0-69.2)	57.2% (62.9-71.5)	66.2% (62.9-69.5)	0.713
<i>Pre-SP, s, median (IQR)</i>	14.3 (9.0-21.0)	13.5 (6.0-18.0)	15.0 (10.9-21.5)	0.121
<i>Post-SP, s, median (IQR)</i>	7.0 (4.0-12.0)	6.0 (3.5-8.2)	8.7 (4.5-13.9)	0.053
<i>PSP, s, median (IQR)</i>	20.0 (15.7-27.0)	18.0 (12.3-24.0)	24.0 (16.7-30.2)	0.022

Table 2.

Shock Pauses and Outcomes by Year

Characteristic	Pre-2010 (n=34)	Post-2010 (n=67)	p-value
<i>RA, % (95% CI)</i>	47.1 (29.4-64.7)	37.3 (25.4-49.2)	0.346
<i>Survival, % (95% CI)</i>	32.4 (15.8-48.9)	26.9 (16.0-37.8)	0.565
<i>PreSP, median (IQR)</i>	15.0 (9-22.5)	14 (8.5-19.8)	0.45
<i>PostSP, median (IQR)</i>	8.6 (5-13)	6.9 (3.5-9.3)	0.141
<i>PSP, median (IQR)</i>	23.2 (15.3-28.5)	20 (15-26.9)	0.651

Table 3.

Multiple Logistic Regression Model Results

Model 1:All SP durations			
Predictor	Odds Ratio	p-value	95% CI
<i>PreSP</i>	0.95	0.064	0.89-1.00
<i>PostSP</i>	0.97	0.346	0.90-1.04
<i>Age</i>	1.02	0.311	0.98-1.05
<i>Sex</i>	1.06	0.906	0.41-2.43
<i>EMS Witnessed</i>	0.4	0.324	0.07-2.46
<i>Post-2010</i>	0.64	0.377	0.24-1.73
<i>Shockable First Rhythm</i>	1.10	0.842	0.42-2.91
Model 2:SP durations of 30 seconds or less			
Predictor	Odds Ratio	p-value	95% CI
<i>PreSP</i>	0.90	0.021	0.82-0.98
<i>PostSP</i>	0.89	0.03	0.79-0.99
<i>Age</i>	1.02	0.255	0.99-1.05
<i>Sex</i>	0.93	0.887	0.33-2.62
<i>EMS Witnessed</i>	0.27	0.193	0.04-1.93
<i>Post-2010</i>	0.57	0.307	0.20-1.67
<i>Shockable First Rhythm</i>	0.87	0.8	0.29-2.61