# ASSOCIATIONS OF CHEST COMPRESSION RELEASE VELOCITY AND AGE, WEIGHT, AND GENDER DURING CARDIAC RESUSCITATION

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**Abstract**

**Background:** Higher chest compression release velocity (CCRV) has been associated with better outcomes after out-of-hospital cardiac arrest (OHCA), and patient factors have been associated with variations in chest wall compliance and compressibility. We evaluated whether patient sex, age, weight, and time in resuscitation were associated with CCRV during pre-hospital resuscitation from OHCA.

**Methods:** Observational study of prospectively collected OHCA quality improvement data in two suburban EMS agencies in Arizona between 10/1/2008 and 12/31/2016. Subject-level mean CCRV during the first 10 minutes of compressions was correlated with categorical variables by the Wilcoxon rank-sum test and with continuous variables by the Spearman's rank correlation coefficient. Generalized estimating equation and linear mixed-effect models were used to study the trend of CCRV over time.

**Results:** During the study period, 2,535 adult OHCA cases were treated. After exclusion criteria, 1,140 cases remained for analysis. Median duration of recorded compressions was 8.70 minutes during the first 10 minutes of CPR. An overall decline in CCRV was observed even after adjusting for compression depth. The subject-level mean CCRV was higher for minutes 0-5 than for minutes 5-10 (mean 347.9 mm/s vs. 339.0 mm/s, 95% CI of the difference -12.4 ~ -5.4, $p < 0.0001$). Males exhibited a greater mean CCRV compared to females [344.4 mm/s (IQR 307.3-384.6) vs. 331.5 mm/s (IQR 285.3-385.5), $p=0.013$]. Mean CCRV was negatively correlated with age and positively correlated with patient weight.

**Conclusion:** CCRV declines significantly over the course of resuscitation. Patient characteristics including male sex, younger age, and increased weight were associated with a higher CCRV.

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## Introduction

Out-of-hospital cardiac arrest (OHCA) continues to be one of the leading public health concerns in the United States with over 359,800 EMS-assessed cases each year.[1] Several CPR quality measures have been independently associated with improved survival and favorable neurological outcomes including chest compression rate, compression depth, compression fraction and pre-shock pause.[1-4] The CPR measure termed chest compression release velocity (CCRV) is defined as the maximum velocity of chest release in the posterior to anterior direction.[5] Kovacs *et al*. and Indik *et al*. both demonstrate independent associations between higher CCRV and improved outcomes.[5,6] While CCRV has been associated with improved outcomes, there is wide variability in mean CCRV during OHCA resuscitations.[5] The sources of variability in CCRV in resuscitation are not well studied.

Multiple studies have shown that chest wall compliance increases significantly with repeated compressions, suggesting that compression dynamics are not constant during the performance of CPR.[7,8] Other studies have demonstrated that the amount of force required to achieve equal compression depth varies significantly between patients.[7,9] These findings suggest that patient-related factors influence compression dynamics and therefore may affect the ability to achieve higher rates of CCRV. Our aim was to evaluate how CCRV changes over the course of resuscitation and to assess associations between CCRV and patient sex, age, and weight.

## Methods

### *Study Design and Population*

This was a retrospective, observational, cohort study of consecutive adult OHCA patients treated by two EMS agencies that participate in the Save Hearts in Arizona Registry and Education (SHARE) program and used minimally interrupted cardiac resuscitation as their adult CPR protocol between 10/1/2008 and 12/31/2016.[10-14] Cases were included for analysis if EMS attempted resuscitation and the case duration was at least one minute long. Cases were excluded if the patient had a do-not-resuscitate order, the cardiac arrest occurred in a healthcare facility, the arrest was witnessed by EMS providers, the arrest was presumed to be non-cardiac in etiology (e.g., respiratory, trauma, or drowning), or if CPR quality data were not available including a minimum of 20 compressions during the first 10 minutes of the resuscitation (Figure 1). Primary outcomes were the rate of decline of mean CCRV over time and a comparison of mean CCRV between minutes 0-10, 0-5, and 5-10. Our secondary analysis assessed associations between mean CCRV and patient sex, age, and weight.

### *Data Collection*

Chest compression quality information, including CCRV, were obtained from defibrillators (E Series and X Series; ZOLL Medical, Chelmsford, MA) equipped with accelerometer-based compression sensing technology. Data were collected from the SHARE program OHCA database which has been described previously.[10] As an Arizona Department of Health Services (ADHS)-sponsored public health initiative, Arizona's Attorney General has determined that the SHARE Program is exempt from the requirements of the Health Insurance Portability and Accountability Act (HIPAA), allowing linkage of EMS and hospital data, tracking of OHCA events, and evaluation of efforts to improve resuscitation care. As a public health initiative, the ADHS Human Subjects Review Board and the University of Arizona Institutional Review Board (IRB) have determined that neither the interventions nor their evaluation constitute Human Subjects Research and have approved the publication of de-identified data. The SHARE Program is registered at clinical Trials.gov#NCT01999036.

*Statistical Methods*

The subject-level mean of CCRV was calculated for the time interval of the first 10 minutes, then separately for the first five minutes and the second five minutes. The subject-level mean CCRV was compared between males and females and across quartiles of age and weight using the Wilcoxon rank-sum test, and was also correlated with age and weight as continuous variables using the Spearman's rank correlation coefficient (Spearman's rho), with the rho parameter tested against 0. Mean CCRV levels in the first and second 5-minute intervals were compared using the robust standard error of the difference obtained from generalized estimating equations (GEE) to account for intra-person correlation. Mixed effect models were applied to compression-level data to study the trend of CCRV over time, with or without adjustment for the depth of each compression. A non-linear function of CCRV over time was estimated non-parametrically using penalized thin plate regression splines through the generalized additive model.[15] A random intercept for each subject was included in the model to account for potential correlation of multiple measurements over time from the same subject. All tests were two-sided with significance levels of $p<0.05$. The statistical environment R was used for all analyses.[16]

## Results

A total of 2,535 adult OHCA cases were treated during the study period. After exclusion criteria were applied, 1,140 cases remained for analysis (Figure 1).

The analysis focused on the first 10 minutes of CPR. The 1,140 cases received a total of 852,963 compressions with a median of 790 compressions per subject (IQR 689.5, 858.0). The median duration of recorded compressions was 8.70 minutes (IQR 8.0, 9.1) per subject. The mean CCRV was 343.3 mm/s for the first ten minutes of CPR. As shown in table 1, the mean CCRV was higher for minutes 0-5 than for minutes 5-10 (mean 347.9 mm/s vs. 339.0 mm/s; decrease in the mean = 8.9 with 95% CI 5.4~12.4 from GEE with $p < 0.0001$). CCRV declined over time with a rapid decrease in the first two minutes of resuscitation, even after adjusting for compression depth (Figure 2).

Male patients exhibited a greater overall subject-level mean CCRV compared to female patients (Table 2). The mean CCRV was negatively correlated with age (older subjects having lower mean CCRV) and positively correlated with weight (heavier subjects having higher mean CCRV). All p-values were < 0.0001. Both sex and age independently were still strongly correlated with CCRV after adjusting for compression depth (both p-values < 0.0001; Table 3). The association between patient weight and CCRV, however, disappeared after controlling for compression depth ($p > 0.99$).

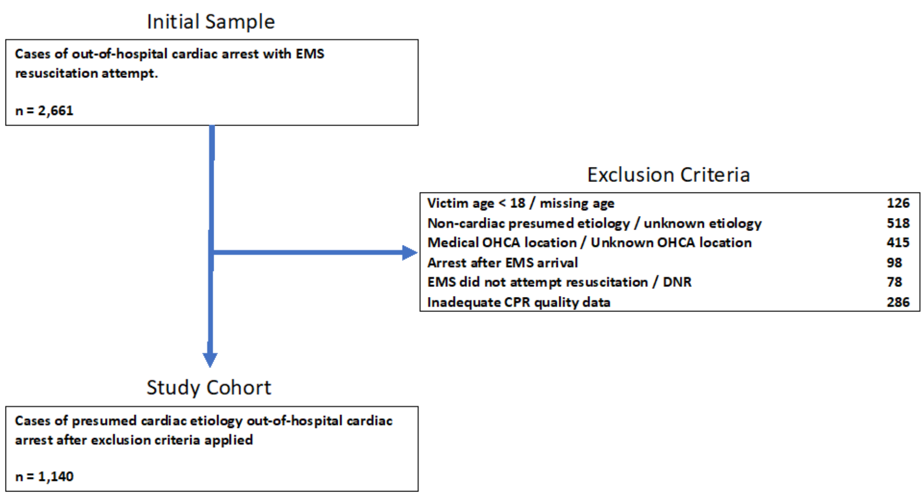


*Figure 1. Exclusion criteria and study population*

| | Mean (Standard deviation; mm/s) | Median (mm/s) | IQR (mm/s) | Range (mm/s) |
|---|---|---|---|---|
| 0-5 min | 347.9 (77.1) | 344.6 | (297.3, 394.8) | (118.4, 610.6) |
| 5-10 min | 339.0 (76.9) | 338.7 | (289.3, 383.8) | (67.6, 732.5) |
| 0-10 min | 343.3 (71.0) | 341.7 | (298.2, 384.8) | (81.7, 627.2) |

*Table 1. Summary of subject-level CCRV means in different time intervals*

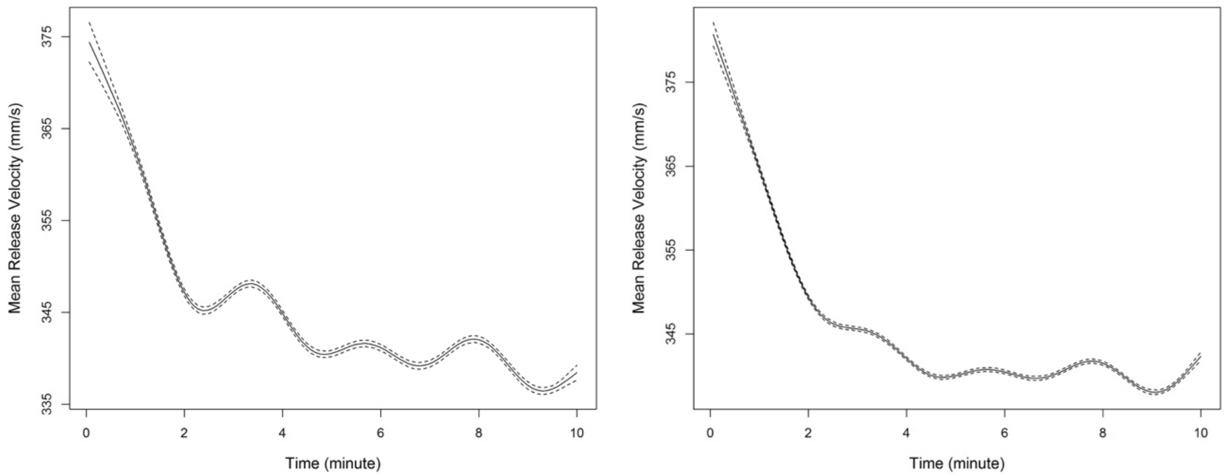


*Figure 2. Mean chest compression release velocity over time, without adjustment for compression depth (left); mean chest compression release velocity over time adjusted for depth (right). Pointwise 95% confidence bands were shown by dotted curves.*

*Table 2. Associations between subject-level mean chest compression release velocity and victim sex, age and weight.*

| Variable | Summary[a] | | Subject-level Mean CCRV (mm/s)[b] | p-value[c] |
|---|---|---|---|---|
| Sex | Female | 385 (33.8%) | 331.5 (285.3, 385.5) | 0.0133 |
| | Male | 755 (66.2%) | 344.4 (307.3, 384.6) | |
| Age (year) | 65 (54, 76) | | -0.226 | < 0.0001 |
| Age quartile | [18,54] | 300 (26.3%) | 364.4 (323.1, 407.5) | < 0.0001 |
| | (54,65] | 286 (25.1%) | 341.4 (300.4, 388.6) | |
| | (65,76] | 272 (23.9%) | 340.3 (298.2, 379.1) | |
| | (76,104] | 282 (24.7%) | 315.8 (282.5, 362.6) | |
| Weight (kg) | 89.5 (73.0, 105.0) | | 0.239 | < 0.0001 |
| Weight quartile | [32,73] | 234 (20.5%) | 316.4 (272.6, 367.7) | < 0.0001 |
| | (73,89.5] | 188 (16.5%) | 342.8 (308.4, 388.2) | |
| | (89.5,105] | 213 (18.7%) | 354.2 (312.8, 395.1) | |
| | (105,273] | 209 (18.3%) | 360.2 (317.3, 409.8) | |
| | Unknown | 296 (26%) | 334 (295.4, 376.8) | |

[a] Count (percentage) for categorical variables, and median (IQR) for continuous variables.

[b] Median (IQR) of subject-level CCRV was shown for categorical variables, and estimated Spearman's rank correlation coefficient (rho) was shown for continuous variables.

[c] For each categorical variable, subject-level mean CCRV was compared across all categories by Wilcoxon rank-sum test; for each continuous variable, it's correlation with subject-level mean CCRV (Spearman's rho) was tested against 0.

*Table 3. Association of release velocity and victim sex, age and weight after adjusting for compression depth and time in CPR*

| Variable | Levels | Effect (mm/s) | 95% CI | p-value |
|---|---|---|---|---|
| Compression depth (mm) | | 5.8 | (5.78, 5.81) | < 0.0001 |
| Age (year) quartile | [18,54] | referent | --- | < 0.0001 |
| | (54,65] | -8.19 | (-13.98, -2.40) | |
| | (65,76] | -15.72 | (-21.59, -9.86) | |
| | (76,104] | -20.29 | (-26.18, -14.40) | |
| Weight (kg) quartile | [32,73] | referent | --- | 0.9973 |
| | (73,89.5] | -0.84 | (-7.83, 6.15) | |
| | (89.5,105] | -0.33 | (-7.09, 6.43) | |
| | (105,273] | -0.96 | (-7.88, 5.96) | |
| | Unknown | 0.06 | (-6.15, 6.27) | |
| Sex | Female | referent | --- | < 0.0001 |
| | Male | 11.07 | (6.57, 15.58) | |
| Time after CPR started (minute) | Nonparametric function | | | < 0.0001 |

## Discussion

Cardiopulmonary resuscitation science is complex and our knowledge of factors that influence patient outcomes continues to evolve. While the assessment of CPR quality measures such as compression rate, depth, and fraction are traditionally rescuer-centered, patient and situation factors (e.g. duration of resuscitation) likely play a role. One of the most recently identified CPR measures is CCRV, which describes the maximum velocity at which the chest wall rises after compression.[4-6] Little is known about how arrest victim characteristics may affect CCRV and how CCRV evolves over the course of resuscitation. To our knowledge, we were the first study to evaluate CCRV over time and to assess any associations between CCRV and the patient factors of sex, age, and weight.

*CCRV Over Time*

Studies to date suggest that higher CCRV is associated with better outcomes; however, the impact that repeated compressions have upon CCRV and how CCRV changes over the course of resuscitation is not documented. In animal models, the generation of intrathoracic vascular pressure during CPR is largely dependent on the initial thoracic anatomy and the change of the initial anatomy as a result of deformities produced during compression.[17] Tomlinson *et al*. demonstrated that a decreased amount of force is required to achieve an equal level of compression depth over time during CPR, suggesting that repeated compressions soften the chest wall.[7] Similarly, Segal *et al.* showed that the compliance of the chest wall increases significantly over time during CPR.[8] Based upon these studies, we hypothesized, and our results suggest, that release velocity declines with repeated compressions. The mean CCRV in our sample was 8.9 mm/s higher for minutes 0-5 than for minutes 5-10 (Table 1). Additionally, our results demonstrate a sharp decline early in resuscitation even when controlled for depth (Figure 2). This finding, in context of prior literature, suggests that loss of chest elasticity may impact CCRV over the course of resuscitation. While the decline in CCRV could be influenced by provider factors such as fatigue and increased incomplete rescuer release during recoil, also referred to as “leaning”, we believe these to be minimal due to resuscitation protocols designed to minimize fatigue through practices including rescuer switching and defibrillator feedback.

*CCRV and Patient Characteristics*

Our study found several patient factors that appear to impact CCRV, including patient sex, age, and weight. Variation in chest wall compressibility has been demonstrated between male and female patients. Tomlinson *et al.* described how less force is required to achieve the same level of compression depth in female patients compared to male patients.[7] It was suggested that differences in chest wall anatomy contributed to increased chest wall compliance in female patients. In our study, mean CCRV values were statistically higher in male patients compared to female patients (344.4 mm/s and 331.5 mm/s, respectively p=0.01) (Table 2), suggesting that anatomical differences between men and women may also contribute to the ability of the chest wall to achieve higher release velocities.

In addition to patient sex, there was significant variation in mean CCRV between age groups in our results. After adjusting for depth, we observed the CCRV to be 20.29 mm/s slower in the oldest age quartile compared to the youngest age quartile (Table 3). Several studies have demonstrated that increased age is associated with a decline in hyaline cartilage elasticity, a key component in chest wall recoil.[18] Porcine models have shown significant age-related chest morphology differences during compressions with subsequent differences in the intrathoracic pressures that are generated.[17] In humans, chest compliance has been shown to decline with age and rib cage deformation.[19-21] Our results, in context with the studies above, suggest that differences in chest wall physiology between older and younger victims of OHCA could be a factor contributing to the difference in CCRV observed between these groups.

Our study also suggested that CCRV tends to increase at greater patient weights. The heaviest quartile of victims demonstrated 13.8% higher CCRV than the lowest quartile [316.4 mm/s (272.6, 367.7) vs 360.2 mm/s (317.3, 409.8)]. Increased chest wall resistance and atypical diaphragmatic position are anatomical differences that have been described in obese individuals.[22] As shown in Table 3, statistical differences between sex and age groups were maintained after adjusting for depth. However, the differences among weight quartiles were not significantly different after the adjustment. While it is possible that these anatomical differences in individuals with larger weights impact CCRV, our results suggest that deeper compressions in heavier patients may be impacting CCRV.

*CCRV: Victim vs Rescuer Dependent Factors*

Current guidelines recommend that the rescuer allow for complete chest wall recoil between compressions during CPR.[23] The ability of the chest wall to recoil is clearly impacted by the presence of rescuer leaning over the patient's chest, impeding full chest expansion.[24] In animal studies, leaning has been shown to decrease coronary perfusion pressure, cardiac output, and myocardial blood flow.[25] As CCRV is the maximum speed of recoil of the chest, CCRV may be impacted by incomplete recoil due to rescuer leaning. While rescuer-fatigue and leaning are described as likely causes of CCRV variance in previous studies, our results are the first to suggest that CCRV may be impacted by patient factors and duration of resuscitation in addition to rescuer-dependent elements.[4-6]

## Conclusion

Chest compression release velocity declines over time during prehospital cardiopulmonary resuscitation and is associated with patient sex, age, and weight. Male sex, younger age, and increased weight were associated with higher CCRV. Further research is necessary to elucidate how to optimize the CCRV over the course of resuscitation with these factors in mind.

## Limitations

The EMS systems studied in this analysis were involved in a dedicated quality assurance system making the applicability of our findings to other EMS systems without similar optimization efforts uncertain. The measurement of CCRV by the accelerometer may be affected by the release of other compressible surfaces (beds or EMS stretchers) beneath the patient. However, we believe the vast majority of the CPR provided by our EMS agencies occurred on a firm surface (backboard or ground) minimizing the potential impact on our CCRV data. CPR quality data were missing for 286 cases and we excluded these cases from the main analyses. Variations in rescuer performance may have contributed to the observed differences in CCRV, however, the rescuers in this study were equipped with defibrillators with real-time feedback, minimizing inter-rescuer variability. Lastly, our study is observational and, as such, causality cannot be inferred from our study's findings.

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