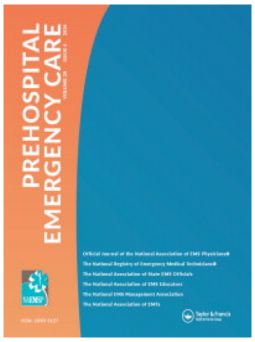

Prehospital Emergency Care

ISSN: (Print) (Online) Journal homepage: www.tandfonline.com/journals/ipec20

Chest Decompressions - the Driver of CPR Efficacy: Exploring the relationship between compression rate, depth, recoil velocity, and end-tidal CO2

Kira Chandran, Isabel M. AlgazeGonzalez, Sixian Wang & Daniel P. Davis

To cite this article: Kira Chandran, Isabel M. AlgazeGonzalez, Sixian Wang & Daniel P. Davis (03 Jun 2024): Chest Decompressions - the Driver of CPR Efficacy: Exploring the relationship between compression rate, depth, recoil velocity, and end-tidal CO2, Prehospital Emergency Care, DOI: 10.1080/10903127.2024.2364058

To link to this article: https://doi.org/10.1080/10903127.2024.2364058

Accepted author version posted online: 03 Jun 2024.

Chest Decompressions - the Driver of CPR Efficacy: Exploring the relationship between compression rate, depth, recoil velocity, and end-tidal CO2

Kira Chandran[a,b], M.D., Isabel M. Algaze Gonzalez[c], FAWM, M.D., Sixian Wang[a], M.D., Daniel P. Davis[d,*], M.D.

[a]Georgetown University School of Medicine, 3900 Reservoir Rd, Washington DC 20007
[b]Harvard Affiliated Emergency Medicine Residency Program, Mass General Brigham, 55 Fruit St, Boston 02114
[c]University of California, Irvine, 1001 Health Sciences Rd, Irvine, CA 92617
[d]Logan Health EMS Division, 310 Sunnyview Lane, Kalispell, MT 59901

***Corresponding Author:** Daniel Davis, M.D., Logan Health EMS Division, 310 Sunnyview Lane, Kalispell, MT 59901, danieldavismd@gmail.com

ABSTRACT

OBJECTIVES: Cardiopulmonary arrest survival is dependent on optimization of perfusion via high quality cardiopulmonary resuscitation (CPR), defined by a complex dynamic between rate, depth, and recoil velocity. Here we explore the interaction between these metrics and create a model that explores the impact of these variables on compression efficacy.

METHODS: This study was performed in a large urban/suburban fire-based emergency medical services (EMS) system over a 9-month period from 2019-2020. Manual chest compression parameters [rate/depth/recoil velocity] from a cohort of out-of-hospital cardiac arrest (OOHCA) victims were abstracted from monitor defibrillators (ZOLL X-series) and end-tidal carbon dioxide (ETCO2) from sensors. The mean values of these parameters were modeled against each other using multiple regression and structural equation modeling with ETCO2 as a dependent variable.

RESULTS: Data from a total of 335 patients were analyzed. Strong linear relationships were observed between compression depth/recoil velocity ($r=0.87$, $p<0.001$), ETCO2/depth ($r=0.23$,

$p<0.001$) and ETCO2/recoil velocity ($r=0.61$, $p<0.001$). Parabolic relationships were observed between rate/depth ($r=0.39$, $p<0.001$), rate/recoil velocity ($r=0.26$, $p<0.001$), and ETCO2/rate ($r=0.20$, $p=0.003$). Rate, depth, and recoil velocity were modeled as independent variables and ETCO2 as a dependent variable with excellence model performance suggesting the primary driver of stroke volume to be recoil velocity rather than compression depth.

CONCLUSION: We used manual CPR metrics from out of hospital cardiac arrests to model the relationship between CPR metrics. These results consistently support the importance of chest recoil on CPR hemodynamics, suggesting that guidelines for optimal CPR should emphasize the importance of maximum chest recoil.

Keywords: recoil velocity, chest recoil, CPR, end-tidal CO2, out of hospital cardiac arrest, structural equation modeling

INTRODUCTION

Cardiopulmonary arrest takes the lives of hundreds of thousands of people each year (1). Survival is dependent upon optimal resuscitation including high quality cardiopulmonary resuscitation (CPR) and early defibrillation (2, 3). The importance of exceeding and maintaining coronary perfusion pressure with high quality CPR cannot be overstated (4-7). End-tidal CO2 (ETCO2) has been shown to correlate with coronary perfusion pressure and cardiac output during CPR, as a way of assessing blood flow during CPR (8). Although CPR guidelines and training have emphasized the importance of optimal chest compressions for several decades, performance remains poor, which may explain the persistence of mediocre survival rates (9-12).

Conceptually, cardiac output with chest compressions should be a function of stroke volume and heart rate, just as with spontaneous perfusion, with compression rate as a proxy for heart rate and stroke volume as the difference in cardiac volume between chest recoil and subsequent compression. While this would appear to justify the traditional axiom of "Push fast and deep", there appears to be a trade-off between fast compression rates and suboptimal compression depth and recoil. Optimal chest compressions are associated with alternating positive and negative intrathoracic pressure, representing a formula for maximal cardiac output. The Resuscitation Outcomes Consortium demonstrated diminished survival with excessively fast compression rates, which appeared to compromise compression depth (13). Unlike spontaneous perfusion, cardiac filling during chest compressions is dependent not only on central venous pressure but also on negative intrathoracic pressure during chest recoil (14). Previous investigations observed that excessively fast rates were associated with incomplete decompression, limiting the negative intrathoracic pressure required for cardiac filling (13). Recoil velocity appears to be an independent predictor of outcomes in cardiac arrest victims (15, 16). Several randomized prospective trials demonstrated improved long-term survival and favorable neurologic outcomes with use of active chest compression-decompression resuscitation (17, 18). However this remains a debated topic with other trials and meta-analysis showing no difference in long term survival and neurologic outcomes (19, 20).

Few studies have explored the relationship between compression depth, rate, recoil, and ETCO2, with notable gaps in the understanding of interactions between compression parameters (21-23). Here we utilize a large database of chest compression metrics to explore these four variables in manual CPR performed on out-of-hospital cardiac arrest victims. We hypothesized the existence of a non-linear relationship between these variables, using ETCO2 as a surrogate for cardiac output.

METHODS

Design

We performed a retrospective analysis utilizing a large CPR database. All cases represented out-of-hospital cardiopulmonary arrest victims from Riverside County collected from November 2019 through August 2020. No patient identifiers are maintained in the database. Thus, waiver of informed consent was granted from the UC Irvine Institutional Review Board.

Setting

Riverside County includes a population of 2.4 million in an area of 7,300 square miles. While the majority of the population lives in an urban environment, the service area also includes suburban, rural, and remote areas as well. Riverside County Fire Department responds to 911 calls from 101 different fire stations, the majority of which provide advanced life support (ALS) first response. The vast majority of first response vehicles include both emergency medical technician - defibrillation (EMT-D) and paramedics. A private ALS transport unit also responds to 911 calls and participates in arrest resuscitation events. During the study period, all first responding EMS providers were trained using the Advanced Resuscitation Training (ART) program, which promotes high-performance CPR as well as the use of ETCO2 to modify target CPR parameters to maximize cardiac output (24). Of note, chest recoil is emphasized by promoting "oil pump" compressions, which involve both hands fully open with fingertips in continuous contact with the chest and complete separation of the thenar/hypothenar eminences from the chest with each compression.

First responding units apply an ALS defibrillator (ZOLL X Series™, ZOLL Medical Corp, Chelmsford, MA), which records chest compression rate, depth (amplitude), and recoil velocity using an accelerometer placed over the sternum. An ETCO2 sensor is placed between the bag-valve apparatus and either the mask or the advanced airway immediately upon initiation of chest compressions. Defibrillators display the cardiac rhythm continuously, including the use of a filter to reduce compression artifact. In addition, chest compression parameters [rate in compressions/min, depth (amplitude) in inches, recoil velocity as a semi-quantitative bar graph] are displayed as real-time feedback. Following case completion, data files are uploaded to a cloud-based repository for performance improvement review by trained nurses and the medical director. File names are annotated to reflect whether patients survived to ED admission.

Subjects

During the nine-month study period (November 2019 - August 2020), CPR data for each cardiopulmonary arrest victim were uploaded onto a secure, web-based server for performance improvement analysis. For this analysis, all adult subjects (age >18 years) with intact data files and a minimum of 15 minutes of CPR were included. Although we recommend the use of capnometry with both bag-valve-mask and advanced airways, the end-tidal CO2 values utilized for this analysis were limited to those obtained following endotracheal intubation. Subjects were excluded if the CPR duration was <15 minutes and for incomplete CPR data [depth, rate, recoil velocity, end-tidal CO2].

Data analysis

Files were deidentified for the purposes of this analysis, with the following variables available for analysis: chest compression duration; chest compression depth, rate, and recoil velocity; end-tidal CO2 (ETCO2) for each minute; number of defibrillation attempts; return of spontaneous circulation, and survival to ED admission. To account for the variability in CPR duration across patients, mean values for each chest compression parameter [depth, recoil velocity, rate] as calculated by the CPR analysis software (Code Review, ZOLL Medical, Chelmsford, MA) were used for all analyses. In addition, a mean value for intra-arrest ETCO2 was calculated for each patient.

The primary goal of this investigation was to explore the interrelationship between chest compression rate, depth, and recoil velocity and their association with ETCO2 as a surrogate for cardiac output. This was accomplished using several different analytical approaches. First, polynomial regression was used to model the interrelationships between each of the chest compression parameters [depth vs. recoil velocity, depth vs. rate, recoil velocity vs. rate] and between each parameter and ETCO2. Second, stepwise multiple regression was used to generate a combined model of chest compression parameters with ETCO2 as the dependent variable. Finally, two different structural equation models were generated. In the first, all three chest compression parameters [rate, depth, recoil velocity] were used as input variables to predict ETCO2. In the second, chest compression depth and recoil velocity were used as input variables for stroke volume, with chest compression rate and stroke volume used as input variables for cardiac output as defined by end-tidal CO2. StatsDirect (Leeds, UK) was used for polynomial regression analysis, and JASP 0.16.4 (University of Amsterdam, Netherlands) was used for stepwise multiple linear regression and structural equation modeling. Statistical significance was assumed for $p<0.05$. Regression coefficients and parameter estimates were used to characterize the relationships between covariables. Structural equation model fit was quantified using Comparative Fit Index (CFI) and Bentler-Bonett Normed Fit Index (NFI), with target values >0.950.

RESULTS

Data from a total of 335 cardiopulmonary arrest victims were analyzed. Mean values for chest compression depth, recoil velocity, rate, and arrest duration as well as mean ETCO2 values for all patients are displayed in Table 1. It is worth noting that we reported the proportion of patients in whom at least one defibrillation attempt was made at any time during resuscitative efforts, since initial rhythm was not available. For this analysis, ROSC was defined by sustained end-tidal CO2 levels for at least 2 minutes in the absence of chest compressions. A strong linear relationship was observed between deeper compressions and higher recoil velocity ($r=0.87$, $p<0.001$). Parabolic relationships were observed between compression rate and both compression depth ($r=0.39$, $p<0.001$) and recoil velocity ($r=0.26$, $p<0.001$), with both faster and slower compression rates associated with shallower compression depth and reduced recoil velocities. These data are displayed graphically in Figures 1a, 1b, 1c.

Linear relationships were observed between ETCO2 and both chest compression depth ($r=0.23$, $p<0.001$) and recoil velocity ($r=0.61$, $p<0.001$), with deeper compressions and higher recoil velocities associated with higher ETCO2 values. Again, a parabolic relationship was observed between compression rate and ETCO2 ($r=0.20$, $p=0.003$), with both faster and slower compression rates associated with lower ETCO2 values. These data are displayed graphically in Figures 2a, 2b, 2c.

Stepwise multiple linear regression modeling using chest compression depth, recoil velocity and compression rate as input variables and end-tidal CO2 as the dependent variable resulted in a moderate predictive ability ($r=0.52$, $p<0.001$). However, the model dropped compression depth due to the covariability with recoil velocity, which was more strongly associated with ETCO2. Due to the non-linear relationship between compression rate and ETCO2, the binomial equation was used in place of compression rate in a second stepwise multiple linear regression model, without improvement in predictive ability ($r=0.51$, $p<0.001$) and again with the model dropping compression depth.

Two structural equation models were generated, each with excellent model performance [CFI>0.95, NFI>0.95]. The first model included each of the three chest compression parameters [rate, depth, recoil velocity] as direct inputs and cardiac output [defined by ETCO2] as the output variable. Higher recoil velocity was moderately associated with higher ETCO2, increased compression depth was weakly associated with higher ETCO2, and faster compression rate was weakly associated with lower ETCO2 (Figure 3). The second model included chest compression depth and recoil velocity as input variables to stroke volume, with compression rate and stroke volume and direct inputs to cardiac output (defined by ETCO2) as the output variable. Stroke volume was moderately associated with higher ETCO2 values, and faster compression rate was weakly associated with lower ETCO2 values. Stroke volume appeared to be primarily influenced by recoil velocity rather than compression depth (Figure 4).

DISCUSSION

We utilized a large database of CPR parameters obtained in out-of-hospital cardiac arrest victims to explore the complex relationship between chest compression rate, depth, and recoil velocity and how they influence cardiac output as reflected by ETCO2. Both stepwise linear regression and structural equation modeling suggested a strong relationship between recoil velocity and ETCO2. In fact, the models prioritized recoil velocity over compression depth, likely due to the strong positive linear correlation between these two variables. Compression rate did not appear to be an important contributor to cardiac output as reflected by ETCO2 but instead displayed a narrow range in which recoil velocity and compression depth were optimized. These data suggest that recoil velocity is more important as a contributor to cardiac output than previously recognized.

The fundamental equation for cardiac output in a perfusing patient incorporates both rate and stroke volume as positive contributors. Although somewhat unconventional, it is useful to consider the physiology of cardiac arrest using this same framework, with cardiac volumes during chest recoil and following compression as correlates to diastolic and systolic volume, respectively. Each of the stepwise regression and structural equation models documented that chest recoil was most strongly associated with cardiac output as reflected by ETCO2 values. This may indicate that cardiac filling is a primary driver of chest compression efficiency. However, it may also suggest that current chest compression performance has more variability in recoil velocity than compression depth. Either interpretation would suggest that CPR training should emphasize full chest recoil to a greater degree than simply instructing providers to “avoid leaning on the chest”.

These data are consistent with the findings of Roos et al as part of a mixed-effect model, with increased ETCO2 values associated with deeper compressions, slower ventilation rates, and faster recoil velocities (25). In addition, clinical investigations document an association between chest compression recoil velocity >400 mm/s and increased survival and improved neurologic outcomes (15, 16). In addition, animal studies have associated incomplete chest recoil and leaning with reduced cardiac output (14). This supports previous literature about active compression-decompression devices which are thought to increase negative intrathoracic pressure during chest decompressions via external mechanism (17). Similarly, impedance threshold devices allow for improved chest recoil by restricting inflow of air to the lungs during chest decompression. Although outcomes of active compression-decompression devices remain inconclusive, it does highlight the importance of chest recoil as a variable in compressions (17-20). Kovacs et al. observed that recoil velocity is not independent from other CPR parameters (15). Our study is the first to model compression rate, depth, and recoil velocity with ETCO2 as a surrogate for cardiac output. The influence of recoil velocity on ETCO2 combined with the strong correlation between recoil velocity and compression depth may suggest that a key role of compression depth is to facilitate faster recoil velocities.

While the cardiac output equation would seem to value faster rates, the adverse effect of rate on both depth and recoil velocity explains the decrease in ETCO2 with faster compressions, which is also true for spontaneous circulation. Somewhat surprising was the relationship between slower compressions and reduced recoil velocity. This may suggest that maximizing recoil requires the absence of contact with the sternum during decompression, which is part of the rationale for our use of “oil pump” compressions.

The ILCOR guidelines currently recommend a narrow range for depth (50-60 mm) and rate (100-120) (25). However, in most studies a relatively small percentage of patients receive compressions >50 mm in depth. The Resuscitation Outcomes Consortium observed a relationship between increased compression depth and improved survival, with a recommended maximum compression depth of 40.3-55.3 mm (2, 26). Again, only a small percentage of patients had compression depths recorded above the recommended maximum values, making it difficult to draw definitive conclusions about this group. In addition, while female patients appeared to have a survival peak between 2-2.5 inches, male patients did not demonstrate a decline in outcomes with deeper compressions (26).

There are multiple limitations that must be considered when interpreting these data. We did not incorporate clinical outcomes into the analysis, as our primary objective was to model chest compression parameters against ETCO2 as a surrogate for cardiac output. In this context, the present study should be considered physiological and hypothesis generating. The use of ETCO2 as a surrogate marker for cardiac output is justified based on both preclinical and clinical data (8, 27).

LIMITATIONS

The limitations of this analysis must be considered when interpreting these data. Mean values for chest compression rate, depth, and recoil velocity as well as mean ETCO2 during arrest were used for all models. The compression parameters were automatically calculated by the performance improvement software, with neither individual compression data nor minute-by-minute values available for this analysis. This automatically adjusts for the variability in CPR duration across patients, avoiding disproportionate influence of data from patients with longer resuscitation periods. However, we anticipated that this might increase the likelihood of a Type II error, potentially obscuring a true relationship between ETCO2 and each of the chest compression parameters. The strength of the relationship between recoil velocity and ETCO2 despite the use of mean values across the duration of resuscitation was somewhat unexpected but appears to underscore the importance of this chest compression component. The dynamic relationship between chest compression parameters and ETCO2 remains unproven and is fertile ground for future exploration. The data used in this study was not linked to clinical data, including prehospital initial rhythm, which limits the ability to evaluate patient outcomes or even arrest etiology beyond the delivery of direct countershocks at some time during the resuscitation.

The use of mean ETCO2 values for each patient also fails to account for individual differences with regard to baseline ETCO2 and optimal chest compression dynamics. Variability in thoracic cavity size, the physical properties of each patient's cardiovascular system, and potential fatigue to chest wall structures likely all contribute to defining an optimal set of parameters for compression rate, depth, and chest recoil. Even more important is the potential variability in initial ETCO2 values based on arrest etiology, ventilation, temperature suggesting that dynamic changes in ETCO2 may be more useful than absolute ETCO2 to indicate relative increases and decreases in cardiac output (28-30). We did not incorporate ventilation rate into the models due to the difficulties in accurately measuring this variable during CPR. Providers apply a single breath on the upstroke of every tenth compression, which should limit the incidence of inadvertent hyperventilation, which has potential to reduce ETCO2 values by decreasing PaCO2 values and by decreasing cardiac output (31, 32). Previous studies have also shown that inadequate ventilation via BVM occurs with upwards of 50% of attempted ventilations, which may contribute to elevated ETCO2 levels during cardiac arrest resuscitation (33). Again, these should have increased the chance of Type II error and made it more difficult to find an association between the variables. Thus, we were somewhat surprised at the strength and consistency of association between recoil velocity and ETCO2, particularly using structural equation modeling. Finally, we could not account for chest wall deformation and the resultant impact on recoil velocity, although the relationship between chest wall integrity and cardiac output may be more complex than previously anticipated (34).

CONCLUSIONS

Here we used manual CPR metrics obtained from out-of-hospital cardiac arrest victims to generate both stepwise regression as well as structural equation models to explore the interrelationship between chest compression rate, depth, and recoil velocity in predicting ETCO2 as an indirect surrogate for cardiac output. The models consistently support the influence of chest recoil on CPR hemodynamics, which is critical in improving cardiac arrest outcomes. These data suggest that guidelines for optimal CPR should emphasize the importance of maximal chest recoil.

ACKNOWLEDGEMENTS: The authors would like to thank the paramedics and Emergency Medical Technicians of the Riverside County Fire Department for their excellent service to the citizens of Riverside County.

DECLARATION OF INTEREST STATEMENT: The authors whose names are listed immediately below certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent- licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge or beliefs) in the subject matter or materials discussed in this manuscript.

DECLARATION OF GENERATIVE AI IN SCIENTIFIC WRITING: *"The authors did not use a generative artificial intelligence (AI) tool or service to assist with preparation or editing of this work. The author(s) take full responsibility for the content of this publication."*

Funding Sources: *None*

Disclosure statement: *"The authors report there are no competing interests to declare."*

REFERENCES

1. Tsao CW, Aday AW, Almarzooq ZI, et al.: Heart Disease and Stroke Statistics - 2023 Update: A Report from the American Heart Association. Circulation. 2023;February 21;147(8):E93–621.
2. Nichol G, Daya MR, Morrison LJ, et al.: Compression depth measured by accelerometer vs. outcome in patients with out-of-hospital cardiac arrest. Resuscitation. 2021;October;167:95–104.
3. Vadeboncoeur T, Stolz U, Panchal A, et al.: Chest compression depth and survival in out-of-hospital cardiac arrest. Resuscitation. 2014;February;85(2):182–8.
4. Halperin HR, Lee K, Zviman M, et al.: Outcomes from low versus high-flow cardiopulmonary resuscitation in a swine model of cardiac arrest. Am J Emerg Med. 2010;February 1;28(2):195–202.
5. Kern KB, Ewy GA, Voorhees WD, et al.: Myocardial perfusion pressure: A predictor of 24-hour survival during prolonged cardiac arrest in dogs. Resuscitation. 1988;October 1;16(4):241–50.
6. Naim MY, Sutton RM, Friess SH, et al.: Blood Pressure– and Coronary Perfusion Pressure–Targeted Cardiopulmonary Resuscitation Improves 24-Hour Survival From Ventricular Fibrillation Cardiac Arrest. Crit Care Med. 2016;November;44(11):e1111–7.
7. Paradis NA, Martin GB, Rivers EP, et al.: Coronary Perfusion Pressure and the Return of Spontaneous Circulation in Human Cardiopulmonary Resuscitation. JAMA. 1990;February 23;263(8):1106–13.
8. Falk JL, Rackow EC, Weil MH: End-Tidal Carbon Dioxide Concentration during Cardiopulmonary Resuscitation. N Engl J Med. 1988;March 10;318(10):607–11.
9. Abella BS, Alvarado JP, Myklebust H, et al.: Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. JAMA. 2005;January 19;293(3):305–10.
10. Wik L, Kramer-Johansen J, Myklebust H, et al.: Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. JAMA. 2005;January 19;293(3):299–304.
11. Kramer-Johansen J, Myklebust H, Wik L, et al.: Quality of out-of-hospital cardiopulmonary resuscitation with real time automated feedback: A prospective interventional study. Resuscitation. 2006;71:283–92.
12. Edelson DP, Abella BS, Kramer-Johansen J, et al.: Effects of compression depth and pre-shock pauses predict defibrillation failure during cardiac arrest. Resuscitation. 2006;November;71(2):137–45.

13. Stiell IG, Brown SP, Christenson J, et al.: What is the role of chest compression depth during out-of-hospital cardiac arrest resuscitation?*. Crit Care Med. 2012;April;40(4):1192–8.

14. Zuercher M, Hilwig RW, Ranger-Moore J, et al.: Leaning during chest compressions impairs cardiac output and left ventricular myocardial blood flow in piglet cardiac arrest. Crit Care Med. 2010;April;38(4):1141–6.

15. Kovacs A, Vadeboncoeur TF, Stolz U, et al.: Chest compression release velocity: Association with survival and favorable neurologic outcome after out-of-hospital cardiac arrest. Resuscitation. 2015;July;92:107–14.

16. Indik JH, Conover Z, McGovern M, et al.: Amplitude-spectral area and chest compression release velocity independently predict hospital discharge and good neurological outcome in ventricular fibrillation out-of-hospital cardiac arrest. Resuscitation. 2015;July;92:122–8.

17. Plaisance P, Lurie KG, Vicaut E, et al.: A Comparison of Standard Cardiopulmonary Resuscitation and Active Compression–Decompression Resuscitation for Out-of-Hospital Cardiac Arrest. N Engl J Med. 1999;August 19;341(8):569–75.

18. Aufderheide TP, Frascone RJ, Wayne MA, et al.: Comparative Effectiveness of Standard CPR versus Active Compression Decompression CPR with Augmentation of Negative Intrathoracic Pressure for Treatment of Out-of-Hospital Cardiac Arrest: Results from a Randomized Prospective Study. Lancet. 2011;January 1;377(9762):301.

19. Luo X-R, Zhang H-L, Chen G-J, et al.: Active compression-decompression cardiopulmonary resuscitation (CPR) versus standard CPR for cardiac arrest patients: a meta-analysis. World J Emerg Med. 2013;4(4):266–72.

20. Stiell IG, Hébert PC, Wells GA, et al.: The Ontario Trial of Active Compression-Decompression Cardiopulmonary Resuscitation for In-Hospital and Prehospital Cardiac Arrest. JAMA. 1996;May 8;275(18):1417–23.

21. Considine J, Gazmuri RJ, Perkins GD, et al.: Chest compression components (rate, depth, chest wall recoil and leaning): A scoping review. Resuscitation. 2020;January;146:188–202.

22. González-Otero DM, Russell JK, Ruiz JM, et al.: Association of chest compression and recoil velocities with depth and rate in manual cardiopulmonary resuscitation. Resuscitation. 2019;September;142:119–26.

23. Russell JK, Leturiondo M, González-Otero DM, et al.: Chest compression release and recoil dynamics in prolonged manual cardiopulmonary resuscitation. Resuscitation. 2021;October;167:180–7.

24. Davis DP, Graham PG, Husa RD, et al.: A performance improvement-based resuscitation programme reduces arrest incidence and increases survival from in-hospital cardiac arrest. Resuscitation. 2015;July;92:63–9.

25. Roos E De, Vanwulpen M, Hachimi-Idrissi S: Chest compression release velocity: An independent determinant of end-tidal carbon dioxide in out-of-hospital cardiac arrest. Am J Emerg Med. 2022;April;54:71–5.

26. Wyckoff MH, Greif R, Morley PT, et al.: 2022 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science With Treatment Recommendations: Summary From the Basic Life Support; Advanced Life Support; Pediatric Life Support; Neonatal Life Support; Education, Implementation, and Teams; and First Aid Task Forces. Circulation. 2022;December 20;146(25).

27. Stiell IG, Brown SP, Nichol G, et al.: What Is the Optimal Chest Compression Depth During Out-of-Hospital Cardiac Arrest Resuscitation of Adult Patients? Circulation. 2014;November 25;130(22):1962–70.

28. WEIL MH, BISERA J, TREVINO RP, et al.: Cardiac output and end-tidal carbon dioxide. Crit Care Med. 1985;November;13(11):907–9.

29. Sandroni C, Santis P De, D'Arrigo S: Capnography during cardiac arrest. Resuscitation. 2018;November;132:73–7.

30. Grmec Š, Lah K, Tušek-Bunc K: Difference in end-tidal CO 2 between asphyxia cardiac arrest and ventricular fibrillation/pulseless ventricular tachycardia cardiac arrest in the prehospital setting. doi: 10.1186/cc2369 (Epub ahead of print).

31. Heradstveit BE, Sunde K, Sunde G-A, et al.: Factors complicating interpretation of capnography during advanced life support in cardiac arrest—A clinical retrospective study in 575 patients. Resuscitation. 2012;July;83(7):813–8.

32. Gazmuri RJ, Ayoub IM, Radhakrishnan J, et al.: Clinically plausible hyperventilation does not exert adverse hemodynamic effects during CPR but markedly reduces end-tidal PCO 2. Resuscitation. 2012;February 1;83(2):259–64.

33. Idris AH, Aramendi Ecenarro E, Leroux B, et al.: Bag-Valve-Mask Ventilation and Survival From Out-of-Hospital Cardiac Arrest: A Multicenter Study. Circulation. 2023;December 5;148(23):1847–56.

34. Jaeger D, Kalra R, Sebastian P, et al.: Left rib fractures during cardiopulmonary resuscitation are associated with hemodynamic variations in a pig model of cardiac arrest. doi: 10.1016/j.resplu.2023.100429 (Epub ahead of print).

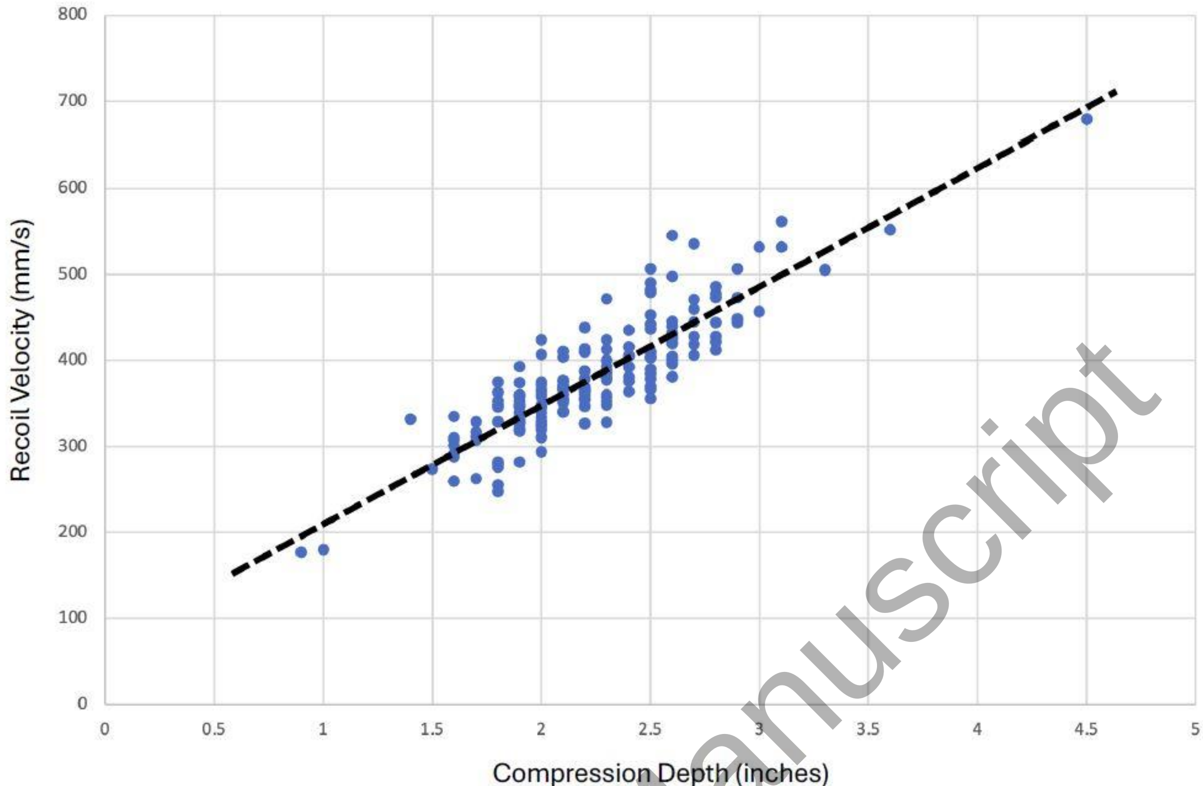

Figure 1a: *Linear regression modeling compression depth against recoil velocity (r=0.87, p<0.001).*

Figure 1a footnotes: *None*

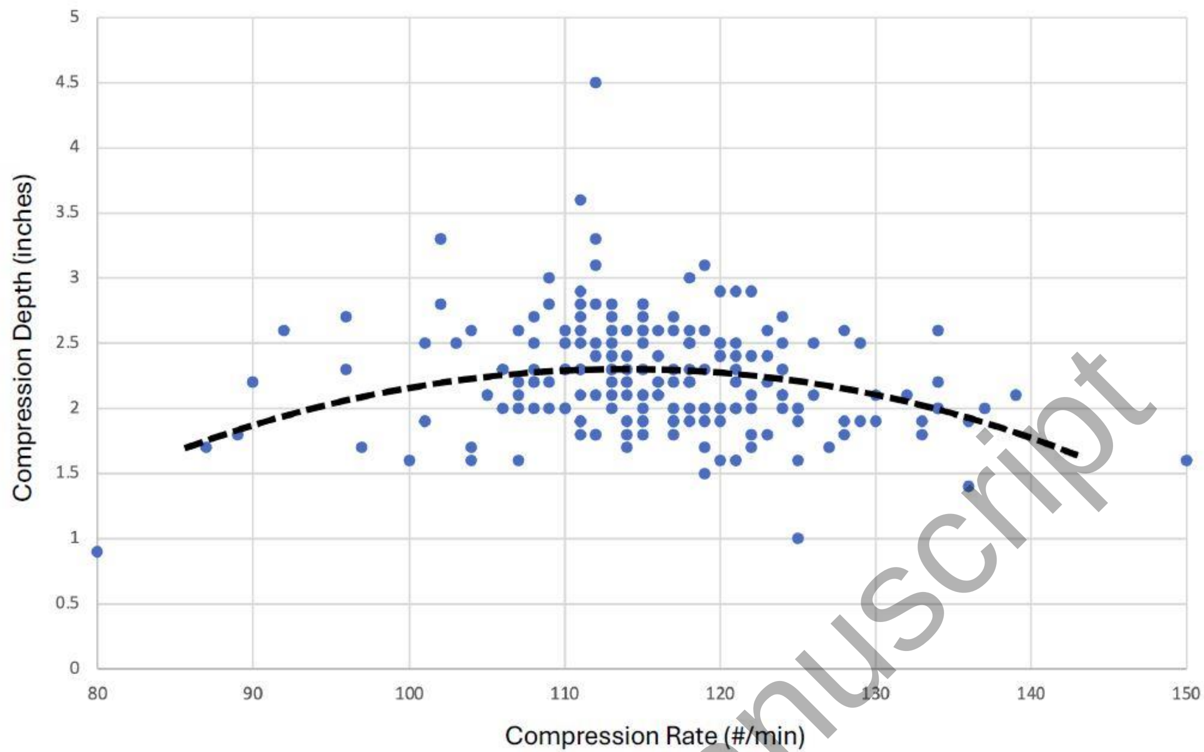

Figure 1b: *Polynomial regression modeling compression rate against compression depth (r=0.39, p<0.001).*

Figure 1b footnotes: *None*

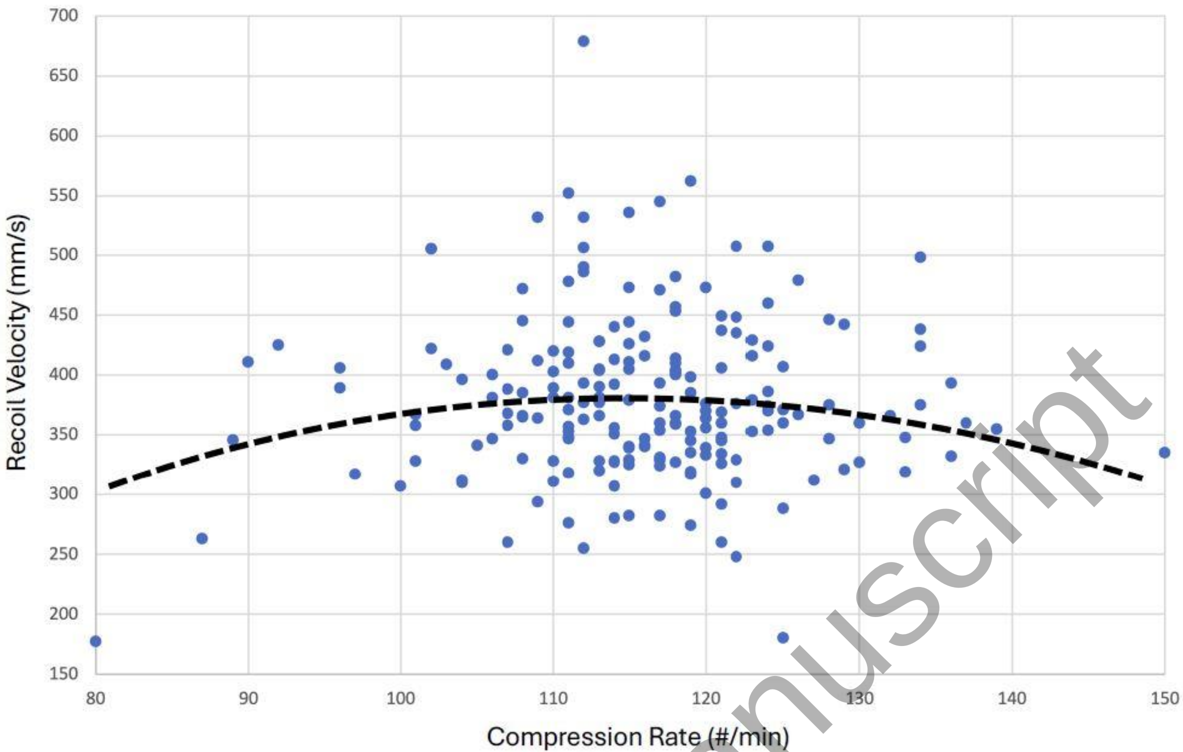

Figure 1c: *Polynomial regression modeling compression rate against recoil velocity (r=0.26, p<0.001).*

Figure 1c footnotes: *None*

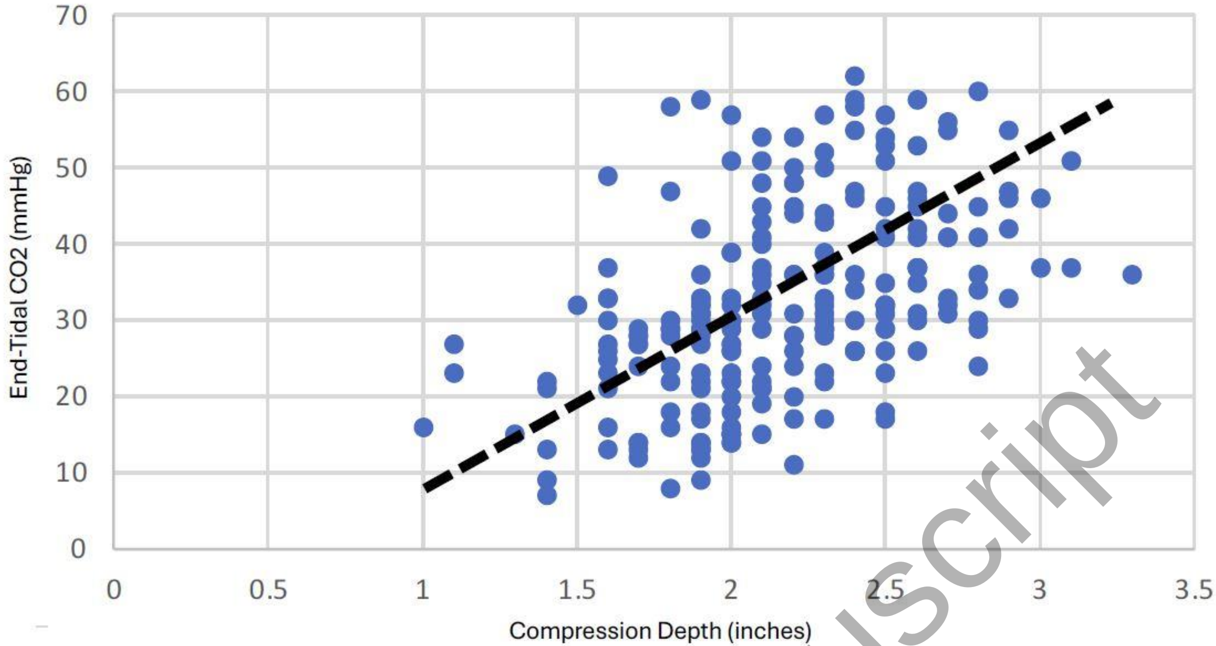

Figure 2a: *Linear regression modeling compression depth against end-tidal CO2 (r=0.23, p<0.001).*

Figure 2a footnotes: *None*

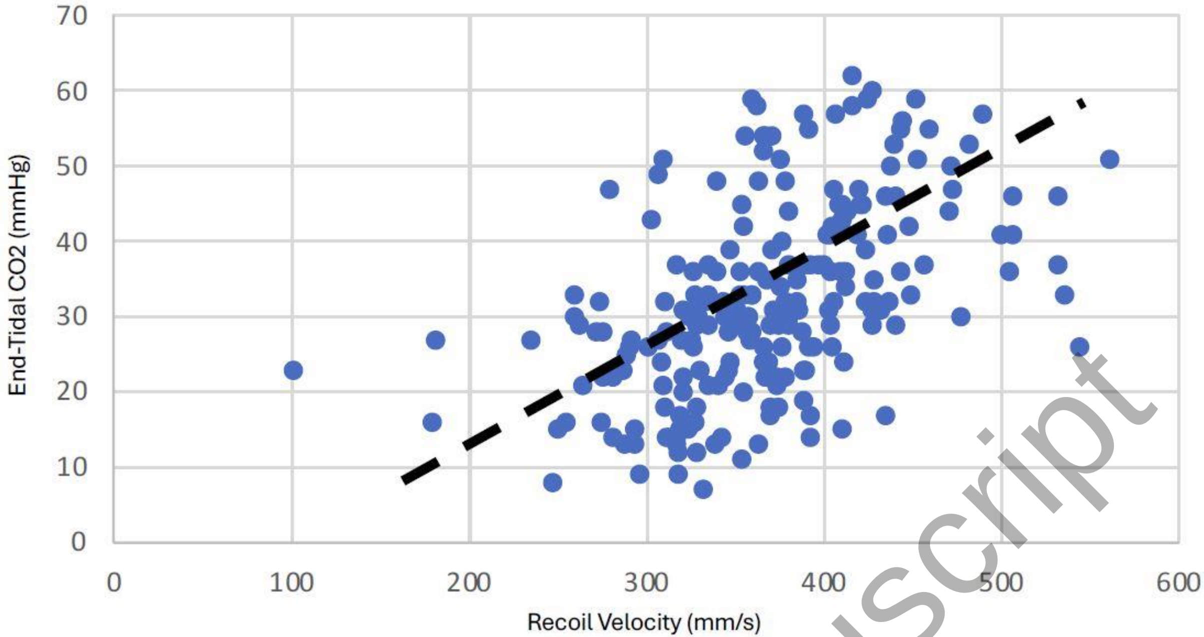

Figure 2b: *Linear regression modeling recoil velocity against end-tidal CO2 (r=0.61, p<0.001).*

Figure 2b footnotes: *None*

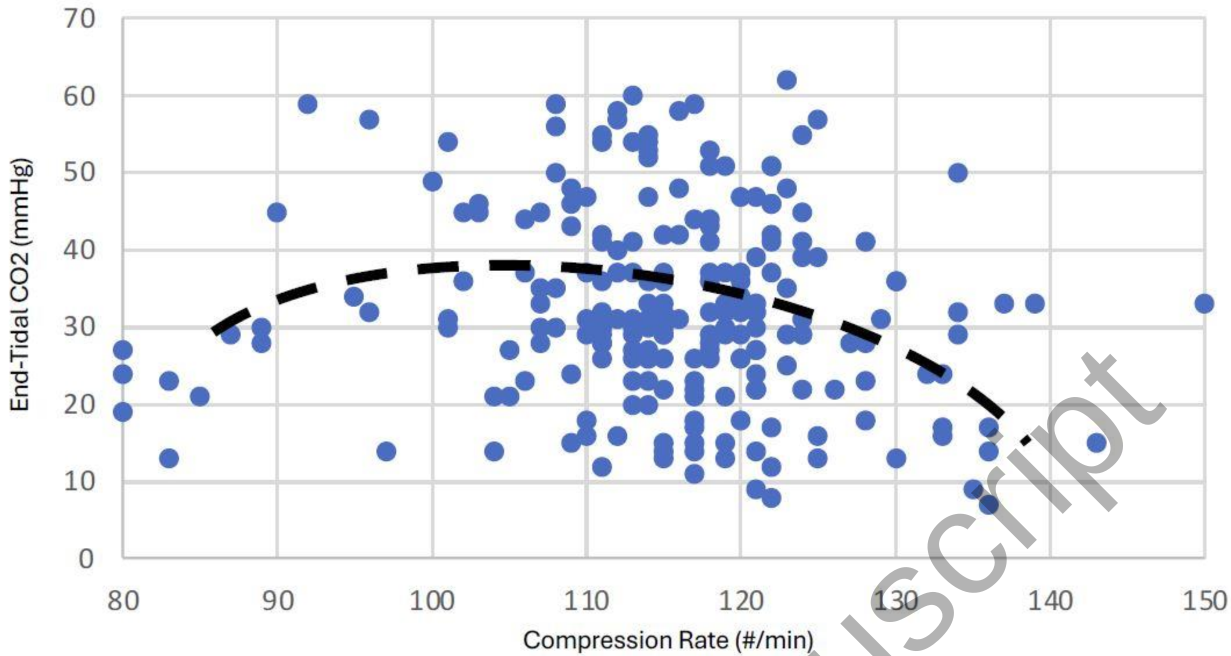

Figure 2c: *Polynomial regression modeling compression rate against end-tidal CO2 (r=0.20, p=0.003).*

Figure 2c footnotes: *None*

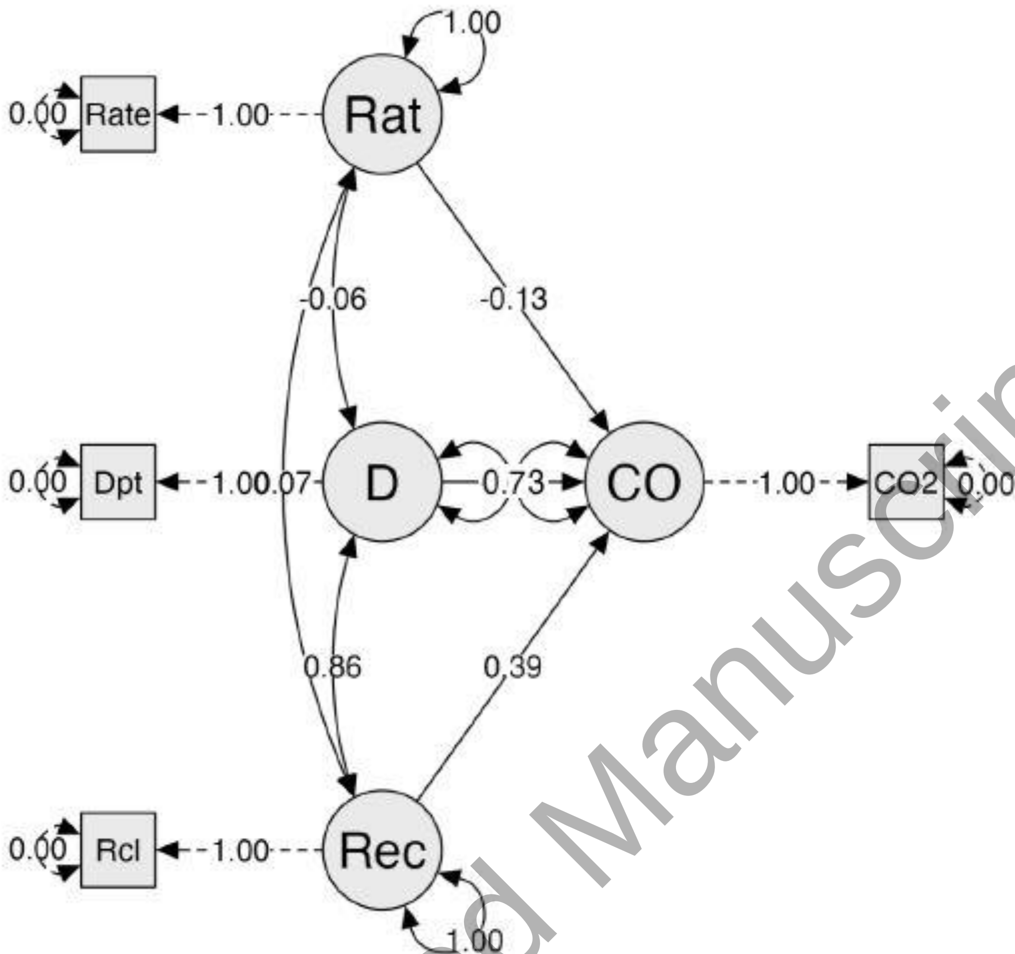

Figure 3: *Structural equation model incorporating compression rate [“Rat” defined by data element “Rate”], compression depth [“D” defined by data element “Dpt”], and recoil velocity [“Rec” defined by data element “Rcl”] as predictors of cardiac output [“CO” defined by data element “CO2”].*

Figure 3 footnotes: *None*

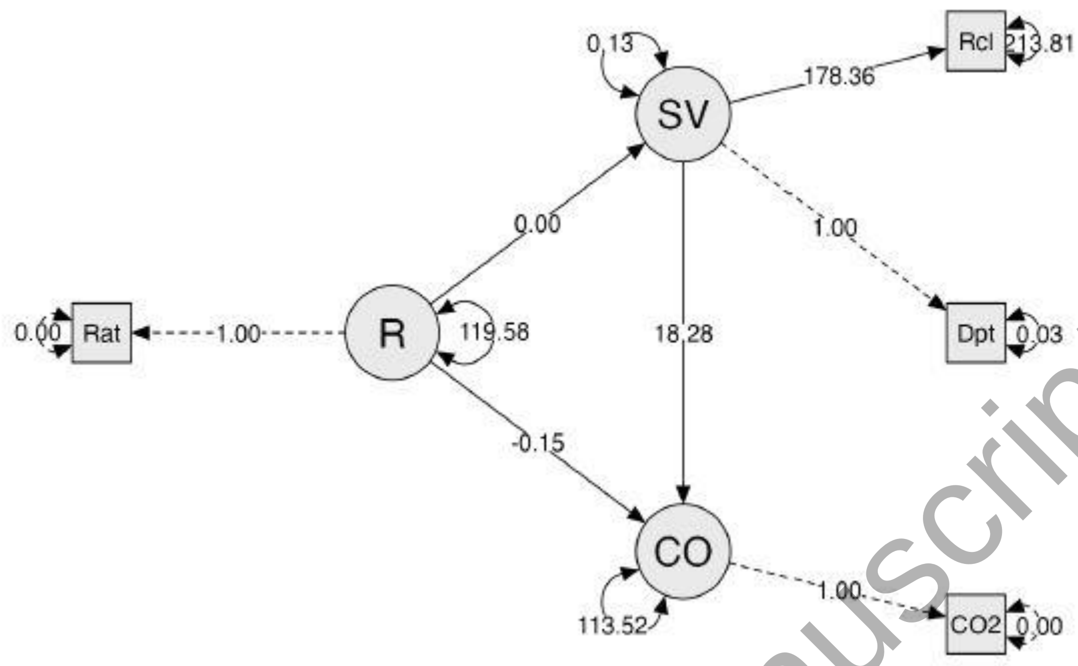

Figure 4: *Structural equation model incorporating compression rate ["R" defined by data element "Rat"] and stroke volume ["SV"] as defined by recoil velocity ["Rcl"] and compression depth ["Dpt"] as predictors of cardiac output ["CO" defined by data element "CO2"].*

Figure 4 footnotes: *None*

Table 1: *Clinical and CPR variables for study population.*

Parameter	Mean or % (95% CI)
CPR duration (min)	22.6 (21.9-23.3)
Depth (inches)	2.2 (2.1-2.3)
Rate (compressions/min)	118 (111-125)
Release velocity (mm/sec)	370 (361-379)
EtCO2 (mmHg)	32.5 (30.9-34.1)
Mean shock (#)	0.7 (0.5-0.9)
Defibrillation attempt during resuscitation (%)	42 (34-50)
Return of spontaneous circulation (%)	42 (36-49)
Survival to ED admission (%)	37 (31-43)

Table 1 footnotes:

CPR = cardiopulmonary resuscitation

CI = confidence intervals

ETCO2 = end-tidal CO2

ED = emergency department