Resuscitation 80 (2009) 458–462

Contents lists available at ScienceDirect

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

Experimental paper

Effect of timing and duration of a single chest compression pause on short-term survival following prolonged ventricular fibrillation☆

Gregory P. Walcott[a,*], Sharon B. Melnick[a,b], Robert G. Walker[b], Isabelle Banville[b], Fred W. Chapman[b], Cheryl R. Killingsworth[a], Raymond E. Ideker[a]

[a] University of Alabama, B140 Volker Hall, 1670 University Blvd., Birmingham, AL 35294, United States
[b] Medtronic Physio-Control, Redmond, WA, United States

ARTICLE INFO

Article history:
Received 17 June 2008
Received in revised form 5 November 2008
Accepted 17 November 2008

Keywords:
Resuscitation
Defibrillation
Sudden cardiac death
Pause

ABSTRACT

Background: Pauses during chest compressions are thought to have a detrimental effect on resuscitation outcome. The Guidelines 2005 have recently eliminated the post-defibrillation pause. Previous animal studies have shown that multiple pauses of increasing duration decrease resuscitation success. We investigated the effect of varying the characteristics of a single pause near defibrillation on resuscitation outcome.
Methods: Part A: 48 swine were anesthetized, fibrillated for 7 min and randomized. Chest compressions were initiated for 90 s followed by defibrillation and then resumption of chest compressions. Four groups were studied—G2000: 40 s pause beginning 20 s before, and ending 20 s after defibrillation, A1: a 20 s pause just before defibrillation, A2: a 20 s pause ending 30 s prior to defibrillation, and group A3: a 10 s pause ending 30 s prior to defibrillation. Part B: 12 swine (Group B) were studied with a protocol identical to Part A but with no pause in chest compressions. Primary endpoint was survival to 4 h.
Results: The survival rate was significantly higher for groups A1, A2, A3, and B (5/12, 7/12, 5/12, and 5/12 survived) than for the G2000 group (0/12, $p < 0.05$). Survival did not differ significantly among groups A1, A2, A3, and B.
Conclusions: These results suggest that the Guidelines 2005 recommendation to omit the post-shock pulse check and immediately resume chest compressions may be an important resuscitation protocol change. However, these results also suggest that clinical maneuvers further altering a single pre-shock chest compression pause provide no additional benefit.

1. Introduction

For sudden cardiac arrest victims with ventricular fibrillation, the two primary treatments available today are rapid defibrillation and performance of chest compressions until the patient's heart can maintain perfusion without help.[1] Automatic external defibrillators (AEDs) have been introduced to extend the use of defibrillators from ambulances into less conventional settings including firefighters, policeman, and lay people.[2] Yet even with their introduction, cardiac arrest survival rates remain very low,[2] possibly because the defibrillator's use has not been optimally coordinated with the delivery of chest compressions.

The new 2005 AHA/ILCOR Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiac Care recommend several protocol changes aimed at reducing hands-off pauses in chest compressions, including elimination of stacked defibrillation shocks and post-shock pulse and rhythm checks.[1] Preliminary clinical data indicate that implementing protocol changes such as these can decrease the AED-prompted hands-off time substantially. This study showed further that these changes decreased the actual amount of hands-off time and were associated with improved resuscitation outcomes.[3] In light of these encouraging findings, it is of interest to determine whether alterations to other chest compression pauses inherent in current protocols, such as not removing hands for shock delivery,[4] might provide further improvement.

There are at least three characteristics of pauses in chest compressions that may be important to a resuscitation effort: (1) the timing of a particular pause with respect to other resuscitation events, (2) the duration of any particular pause, and (3) the total duration of all pauses during a resuscitation. Pauses during resuscitation have been evaluated in several animal studies.[5–8] These studies have not systematically varied these three characteristics to determine their relative importance.

☆ A Spanish translated version of the summary of this article appears as Appendix in the final online version at doi:10.1016/j.resuscitation.2008.11.012.

* Corresponding author. Tel.: +1 205 975 4710; fax: +1 205 975 4720.
E-mail address: gpw@crml.uab.edu (G.P. Walcott).

0300-9572/$ – see front matter © 2008 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.resuscitation.2008.11.012

Understanding whether some chest compression pauses are truly more deleterious than others, based on their location in the resuscitation sequence, is important for further refinement of resuscitation protocols. It is presently unknown whether alteration of the timing or duration of a pre-shock pause meaningfully impacts resuscitation outcomes within a protocol when chest compressions are resumed immediately after delivery of a single shock.

In this study, we used a swine model of prolonged unsupported VF to better elucidate the relationship between resuscitation outcome and the timing and duration of a single chest compression pause early in the resuscitation attempt. Specifically, we hypothesized (1) that removal of the pause associated with post-shock pulse and rhythm checks would improve outcome, and (2) that changes in the length and timing of a single pre-shock pause could further improve outcome.

2. Methods

2.1. Animal preparation

All procedures were approved by the Institutional Animal Care and Use Committee at the University of Alabama at Birmingham. Further, all pre-operative and operative care for animals complied with Section 6 of the Animal Welfare Act of 1989 and adhered to the principles outlined in the "Guide for the care and use of animals," National Institutes of Health publication No. 85-23.

Forty-eight domestic swine, 25–40 kg, were studied in Part A and an additional 12 animals were studied in Part B. Animals were pre-anesthetized with telazol/xylazine (4.4 mg/kg of each) and atropine (0.04 mg/kg), then intubated, anesthetized with isoflurane (1.2–3%) and supported on a pressure-controlled mechanical ventilator (Ohmeda Modulus II, BOC Healthcare, NJ) with a minute ventilation of 10–15 ml/kg/min. Normal saline was administered IV at a rate of 5–10 ml/kg/h. Blood gases and electrolytes were measured every half hour and respiratory parameters and infusion fluid composition were adjusted accordingly. ECG lead II was monitored throughout the study.

The animal was placed in dorsal recumbency. The left and right chest walls were shaved. Self-adhesive defibrillation electrodes were placed on the anterior left and right chest walls. The right jugular vein was isolated and a high fidelity pressure catheter (Millar Microtip, Houston, TX) advanced under fluoroscopy to the junction of the right atrium and superior vena cava. A quadripolar EP catheter was inserted into the left jugular vein and advanced into the apex of the right ventricle for ventricular fibrillation induction. The left carotid artery was isolated and a high fidelity pressure catheter inserted and advanced into the left ventricular cavity. The left femoral artery was isolated and a high fidelity pressure catheter advanced into the descending thoracic aorta. After induction of anesthesia, ventilator oxygen fraction was decreased until the animals' pO_2 was less than 150 mmHg.

2.2. Experimental procedure

All times are referenced to the beginning of ventricular fibrillation. After recording 30 s of baseline data, ventricular fibrillation was induced by applying 60 Hz alternating current to the endocardium of the right ventricle. Fibrillation was allowed to persist unsupported for 7 min, after which ventilation and chest compressions were initiated. Ventilation was performed by restarting the ventilator at the same rate and tidal volume as before ventricular fibrillation induction. Chest compressions were performed using a model 1005 'Thumper' device (Michigan Instruments, Grand Rapids, MI) adjusted to the maximum chest compression depth possible without causing left ventricular pressure to exceed aortic pressure by >10 mmHg. Compression depth was consistent with AHA guidelines for adult chest compressions. Ninety seconds after chest compressions were initiated, they were paused briefly for delivery of a single 200J biphasic defibrillation shock (LIFEPAK® 12 defibrillator/monitor, Medtronic ERS, Redmond, WA).

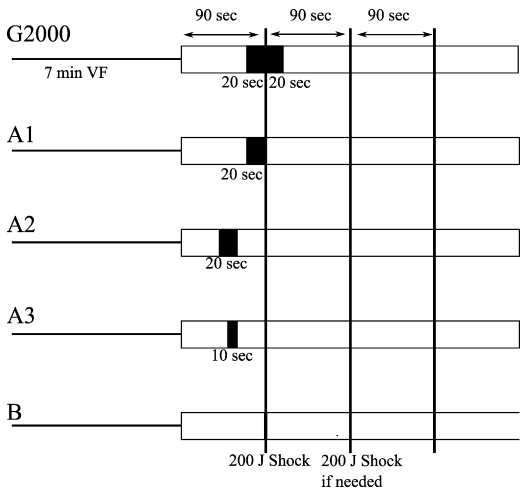

Fig. 1. Timing diagram of resuscitation protocol. Part A: G2000 Group: a 40 s pause starting 20 s before and ending 20 s after attempted defibrillation. Group A1: a 20 s pause just before attempted defibrillation. Group A2: a 20 s pause ending 30 s before attempted defibrillation. Group A3: a 10 s pause ending 30 s before attempted defibrillation. Part B: Group B: no pauses other than the 3 s pause at the defibrillation attempt. Once CPR was resumed after the first defibrillation attempt, the resuscitation protocol was identical for all 5 groups and included a pause for 3 s every 90 s to determine rhythm and perfusion and deliver a defibrillation shock if necessary.

By design, the timing and duration of the pause in chest compressions differed between groups (Fig. 1). In Part A, animals were randomized to four groups. The G2000 group had a pause starting 20 s before and ending 20 s after the first defibrillation shock. This timing was chosen to approximate pause durations required for pre-shock AED rhythm analysis and preparation to administer a shock, plus a post-shock pulse check and preparation to resume chest compressions, typical of well-performed Guidelines 2000-driven care. Groups A1, A2, and A3 each had a pause prior to the first defibrillation shock but the timing and duration of that pause varied with group. These groups were included to evaluate the impact of the characteristics of a pre-shock pause, in the absence of a post-shock pause. In Part B, an additional group of animals (Group B) was studied with an identical protocol, except that the pause in chest compressions was omitted. In all groups, after the first shock, CPR was stopped every 90 s for 3 s to assess heart rhythm, determine whether return of spontaneous circulation (ROSC) had occurred (aortic systolic pressure >50 mmHg), and deliver a single 200J shock if necessary (Fig. 1).

Chest compressions were continued until ROSC occurred or 30 min of resuscitation had elapsed. Following 7.5 min of resuscitation, epinephrine, 0.01 mg/kg, was given every 3 min if ROSC had not occurred, or if aortic systolic blood pressure was less than 50 mmHg. After 1 h, if systolic aortic blood pressure fell below 50 mmHg, dobutamine was started at 5 μg/(kg min) and titrated to the lowest dose that would maintain a systolic aortic blood pressure >100 mmHg. Three hours after fibrillation induction, dobutamine was stopped and the animal was monitored for 1 h. Survival was assessed at 4 h.

Defibrillation success was defined as conversion of a shockable rhythm to a non-shockable rhythm for at least 5 s.[9] Refibrillation was defined as fibrillation recurring more than 5 s after a successful defibrillation shock. Timing of CPR, pauses, and defibrillation was orchestrated via prompts from the automatic slide advance feature of a PowerPoint presentation (Microsoft, Redmond, WA).