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Clinical paper

Effect of compression waveform and resuscitation duration on blood flow and pressure in swine: One waveform does not optimally serve☆

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ABSTRACT

Background: Chest compression (CC) research primarily focuses on finding the 'optimum' compression waveform using a variety of compression efficacy metrics. Blood flow is rarely measured systematically with high fidelity. Using a programmable mechanical chest compression device, we studied the effect of inter-compression pauses in a swine model of cardiac arrest, testing the hypothesis that a single 'optimal' CC waveform exists based on measurements of resulting blood flow.

Methods: Hemodynamics were studied in 9 domestic swine (~30 kg) using multiple flow probes and standard physiological monitoring. After 10 min of ventricular fibrillation, five mechanical chest compression waveforms (5.1 cm, varying inter-compression pauses) were delivered for 2 min each in a semi-random pattern, totaling 50 compression minutes. Linear Mixed Models were used to estimate the effect of compression waveform on hemodynamics.

Results: Blood flow and pressure decayed significantly with time in both arteries and veins. No waveform maximized blood flow in all vessels simultaneously and the waveform generating maximal blood flow in a specific vessel changed over time in all vessels. A flow mismatch between paired arteries and veins, e.g. abdominal aorta and inferior vena cava, also developed over time. The waveform with the slowest rate and shortest duty cycle had the smallest mismatch between flows after about 30 min of CPR.

Conclusions: This data challenges the concept of a single optimal CC waveform. Time dependent physiological response to compressions and no single compression waveform optimizing flow in all vessels indicate that current descriptions of CPR don't reflect patient physiology.

Introduction

Our approach to understanding chest compression generated blood flow has been indirect. While chest compression features such as chest wall mechanics [1], weight [2], downtime [3], rate [4,5], depth [6], non-compression fraction [7], hand position [8], real time caregiver feedback guidance [9,10], and duty cycle [11,12] have been studied, the blood flow generated by chest compressions is rarely measured. Instead metrics include compression rate, compression depth, blood pressures, return of spontaneous circulation (ROSC), or survival to hospital discharge. In the rarer cases when blood flow is measured, it is commonly measured using microsphere deposition in the tissue or a single perivascular flow probe around the carotid artery [13].

As a result, our understanding of the relationship between chest compression mechanics and the resulting hemodynamics is still underdeveloped. Relatively simple questions about blood flow generated by chest compressions have not been addressed experimentally. For example, is a great compression waveform for aortic flow also great for venous return? Does the optimal rate and depth of a compression waveform change as a resuscitation attempt continues? A deeper understanding of how vessels, blood flows and volumes respond to chest compression is important because there are data suggesting that how chest compressions are performed makes a difference in how much blood flow is generated [12,14].

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In this manuscript we report the results of how mechanical chest compressions generated blood flow as a function of time. The experiments were designed to investigate two unspoken assumptions from the resuscitation literature: that there is a single chest compression waveform which is the best for generating and maintaining blood flows, and that chest compression efficacy can be effectively monitored by a single physiological metric, such as the CPP. The physiological measures used in this study are high-fidelity arterial and venous blood flows and pressures reported as a function of the duration of the resuscitation attempt. We investigated the effects of changes in chest compression rate and duty cycle, as might occur as caregivers become tired or simply use other techniques

Methods

The study was approved by the Institutional Animal Care and Use Committee of the Children's Hospital of Philadelphia. All animals received treatment and care in compliance with the 1996 Guide for the Care and Use of Laboratory Animals by the National Research Council in accord with the USDA Animal Welfare Act, PHS Policy, and the American Association for Accreditation of Laboratory Animal Care. All studies were conducted by qualified personnel.

Animal preparation

After an overnight fast, nine female domestic swine (30.7 ± 1.9 kg) were sedated with intramuscular ketamine (20 mg kg^{-1}) and xylazine (2 mg kg^{-1}), followed by inhalation induction of anaesthesia using 4% isoflurane in 100% oxygen. After endotracheal intubation, a surgical plane of anaesthesia was maintained on isoflurane and an inspiratory oxygen fraction of 0.4 in air. The animals were mechanically ventilated with a pressure controlled ventilator (Modulus SE 7900; Datex-Ohmeda Inc., USA) a tidal volume of 12 mL/kg, PEEP 6 cm H_2O, and a rate of 12 breaths/min. The rate was titrated to maintain end-tidal carbon dioxide ($ETCO_2$) of 5.1–5.6 kPa (38–42 mmHg, NM3, Respironics, Philips Healthcare).

After aseptic preparation, solid state pressure transducers (MPC-500, Millar Instruments) were advanced through introducers into the right femoral artery and vein, and placed to measure the aortic pressure (AOP) and the right atrial pressure (RAP), respectively. Pressure probe placement was adjusted to maximize the appearance of the right atrial contraction in the venous pressure tracing and the incisura in the aortic pressure tracing. After surgical exposure, ultrasound blood flow probes were placed around the carotid artery and jugular vein (PS-3, Transonic Systems Inc, USA) and secured with sutures. Via a lateral (right lumbar) laparotomy, ultrasound flow probes were placed around the abdominal aorta, the right renal artery and vein, and the inferior vena cava (Aorta: PAU-10, Renal vessels: PS-2.5, IVC: PAU-12, Transonic Systems Inc, USA). The aortic and IVC flow probes were placed just distal to the renal vasculature. Paralytics were not used, but the surgical plane of anesthesia prevented any observable response to the laparotomy. The laparotomy was closed with a single continuous suture, which allowed ready access to improve the blood flow sensor signal if necessary. Pressure probe and flow probe placement is shown in Fig. 1A. If, at the end of the surgical prep period, the mean arterial pressure was below 60 mmHg, a 20 ml/kg infusion of saline was provided, to offset fluid loss due to fasting and third space losses during surgical preparation.

Compressions were performed mechanically and carefully controlled. Self-adhesive hook and loop fasteners were attached to the chest compressor and the sternum of the animal. This ensured that the sternum and the compressor head were in constant contact during the compression cycle. The compressor head returned to its initial, zero, position at the end of each compression. This results in active return, which is distinct from commercially available active decompression wherein the sternum is pulled to a point above the initial zero position. [15,16] The compressor head was driven by a stepper motor, which allows precise control over motor position. As a result, the compression and relaxation force delivered by the piston varied to meet the waveform descriptions shown in Fig. 1B, and depended on the mechanical properties of the thorax at the time of compression.

Study procedure

After recording baseline values, ventricular fibrillation (VF) was electrically induced and the ventilator was turned off and isoflurane was discontinued for the remainder of the experiment. No muscle relaxers or other paralytics were used during the experiment. After 10 min of untreated VF, two minutes of ramping up compressions were delivered: 100 CPM at 2.5 cm and 100 CPM at 3.8 cm. After the ramping up compressions, the waveform experimentation started.

Compression waveforms were described in terms of five characteristics: depth, compression time, compression hold time, release time, and inter-compression pause time, as shown in Fig. 1B. Duty cycle (DC) expresses the percentage of time being compressed (i.e. the compression time and the compression hold time) as a function of the whole compression cycle. Five distinct chest compression waveforms were performed, with waveform 3 being representative of the current guidelines (S&G) [17]. The waveform parameter values, rate, and duty cycle are shown in Table 1. Chest compression waveforms were delivered for two-minute epochs. We maintained a 15 s pause between epochs simulating rhythm assessment and to allow blood volumes to redistribute as occurs clinically. This pause also minimized the impact of a completed epoch of chest compression effects during the next epoch. Due to the nature of the study, we avoided the use of vasopressors, volume loading or antiarrhythmic medication and did not treat VF if found in the 15-sec pause between waveforms. We made no attempt to reach ROSC in any animal. Ventilations were provided throughout at the tidal volume used before cardiac arrest at a rate of 6 unsynchronized ventilations per minute with 100% O_2 and no PEEP. Positive pressure mechanical ventilation increases the intra-thoracic pressure and alters mechanical chest compression hemodynamics. The crossover design of the experiments, discussed below, allowed for any potential confounding effect of positive pressure ventilation to be spread evenly among the different compression waveforms.

Animals were block randomized to three groups of three each receiving compression waveforms in the order shown in Table 2. The order of the chest compression waveforms was not randomized, but followed five waveform patterns distributing each waveform as evenly as possible across the 25 epochs of CPR.

Data analysis

Data analysis has been previously described. [12] Briefly, the recorded physiological data were divided into two minute CPR epochs by computer, as labeled in Table 2. Within each epoch the data were further segmented into individual compressions. Blood flows were integrated for each compression and then averaged over each epoch, excluding the first 15 s of the epoch. The first 15 s were excluded because they included a transient behavior that reflected a redistribution of blood volume as chest compressions were initiated. This provided the net flow per compression. Minute flows were calculated by multiplying the average net flow by the chest compression rate. To compare the effect of chest compressions as a function of time, CPR epochs were aggregated into 5 epoch cycles, as shown in Table 2. Each cycle contains 5 2-minute epochs of CPR and the 15-second pauses that precede each epoch, totaling 11.25 min of time. Blood flow or pressure results for a given waveform within a cycle represent the mean ± SD for that measure across all three groups delivered at any time within the cycle. All values reported in the manuscript and its figures are net flows. Therefore, discussion of a maximum or minimum flow for a given CPR cycle, is implicitly a discussion of which waveform generated the highest or lowest net blood flow in that CPR cycle.

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