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Leaning during chest compressions impairs cardiac output and left ventricular myocardial blood flow in piglet cardiac arrest

Mathias Zuercher, MD, **Ronald W. Hilwig, DVM, PhD**, **James Ranger-Moore, PhD**, **Jon Nysaether, PhD**, **Vinay M. Nadkarni, MD, FAHA, FCCM**, **Marc D. Berg, MD**, **Karl B. Kern, MD, FACC, FAHA, FACP, FCCP, FSCAI**, **Robert Sutton, MD**, and **Robert A. Berg, MD, FAHA, FCCM**

The University of Arizona Sarver Heart Center (MZ, RWH, MDB, KBK, RAB), Tucson, AZ; Department of Anesthesiology and Intensive Care (MZ), University of Basel, Basel, Switzerland; The University of Arizona Mel and Enid Zuckerman College of Public Health (JR-M), Tucson, AZ; Laerdal Medical (JN), Stavanger, Norway; Department of Anesthesiology and Critical Care Medicine (VMN, RS, RAB), Children's Hospital of Philadelphia and University of Pennsylvania, Philadelphia, PA; and The University of Arizona College of Medicine Steele Children's Research Center and Department of Pediatrics (MDB, RAB), Tucson, AZ.

Abstract

Objective—Complete recoil of the chest wall between chest compressions during cardiopulmonary resuscitation is recommended, because incomplete chest wall recoil from leaning may decrease venous return and thereby decrease blood flow. We evaluated the hemodynamic effect of 10% or 20% lean during piglet cardiopulmonary resuscitation.

Design—Prospective, sequential, controlled experimental animal investigation.

Setting—University research laboratory.

Subjects—Domestic piglets.

Interventions—After induction of ventricular fibrillation, cardiopulmonary resuscitation was provided to ten piglets (10.7 ± 1.2 kg) for 18 mins as six 3-min epochs with no lean, 10% lean, or 20% lean to maintain aortic systolic pressure of 80–90 mm Hg. Because the mean force to attain 80–90 mm Hg was 18 kg in preliminary studies, the equivalent of 10% and 20% lean was provided by use of 1.8- and 3.6-kg weights on the chest.

Measurements and Main Results—Using a linear mixed-effect regression model to control for changes in cardiopulmonary resuscitation hemodynamics over time, mean right atrial diastolic pressure was 9 ± 0.6 mm Hg with no lean, 10 ± 0.3 mm Hg with 10% lean ($p < .01$), and 13 ± 0.3 mm Hg with 20% lean ($p < .01$), resulting in decreased coronary perfusion pressure with leaning. Microsphere-determined cardiac index and left ventricular myocardial blood flow were lower with 10% and 20% leaning throughout the 18 mins of cardiopulmonary resuscitation. Mean cardiac index decreased from 1.9 ± 0.2 $L \cdot M^{-2} \cdot min^{-1}$ with no leaning to 1.6 ± 0.1 $L \cdot M^{-2} \cdot min^{-1}$ with 10% leaning, and 1.4 ± 0.2 $L \cdot M^{-2} \cdot min^{-1}$ with 20% leaning ($p < .05$). The myocardial blood flow decreased from 39 ± 7 $mL \cdot min^{-1} \cdot 100\ g^{-1}$ with no lean to 30 ± 6 $mL \cdot min^{-1} \cdot 100\ g^{-1}$ with 10% leaning and 26 ± 6 $mL \cdot min^{-1} \cdot 100\ g^{-1}$ with 20% leaning ($p < .05$).

Conclusions—Leaning of 10% to 20% (i.e., 1.8–3.6 kg) during cardiopulmonary resuscitation substantially decreased coronary perfusion pressure, cardiac index, and myocardial blood flow.

For information regarding this article, bergra@email.chop.edu.

Keywords

chest compressions; cardiopulmonary resuscitation; cardiac arrest; pediatric; cardiopulmonary resuscitation quality; leaning

Sudden cardiac arrest is a leading cause of death throughout the world, and basic life support with chest compressions is a critical component of the chain of survival for successful resuscitation. The American Heart Association's Guidelines for Emergency Cardiovascular Care and Cardiopulmonary Resuscitation (CPR) recommend allowing complete recoil of the chest wall between chest compressions during CPR (i.e., avoidance of residual leaning on the chest wall) (1, 2). This recommendation is based on the theoretical concept that complete chest recoil can generate negative intrathoracic pressure during CPR, thereby improving venous return and cardiac output.

Limited clinical data indicate that leaning occurs commonly during both adult and pediatric CPR (3, 4). Although there is abundant evidence that the quality of CPR by professional rescuers is typically poor (5–10), data supporting the detrimental effects of residual leaning are sparse. Importantly, there are no data regarding the hemodynamic effects of limited leaning during CPR (e.g., only 10% or 20% of the force used during chest compressions).

This study was designed to determine whether residual leaning adversely affects cardiac index (CI) and left ventricular myocardial blood flow (MBF) in a piglet model of pediatric CPR. We hypothesized that residual leaning at 20% of the force required to maintain aortic systolic pressure ~85 mm Hg would have substantial negative effects on CI and left ventricular MBF but that 10% residual leaning would have minimal or no effect.

MATERIALS AND METHODS

Animal Preparation

This study was conducted with the approval of the University of Arizona Institutional Animal Care and Use Committee. Ten domestic piglets (six female, four male), weighing 10.7 ± 1.2 kg, were anesthetized with 5% isoflurane inhalation anesthetic in oxygen administered by face mask. A cuffed endotracheal tube was placed and a surgical plane of anesthesia was maintained with 1% to 2.5% isoflurane in room air. Mechanical ventilation was delivered at a rate of 10–12/min and an initial tidal volume of 15 mL/kg (Narkomed 2A; North American Drager, Draäger Medical, Telford, PA) and subsequently adjusted to maintain end-tidal carbon dioxide at 40 ± 4 mm Hg measured by an infrared capnometer (No. 47210A; Hewlett-Packard, Palo Alto, CA). The positive end-expiratory pressure was set at 5 cm H_2O.

Animals were placed in dorsal recumbency on the surgical table. Vascular introducer sheaths (Cordis, Miami, FL) were placed in the external jugular vein, a common carotid artery, and both femoral arteries. Solid-state, micromanometer-tipped catheters (MCP-500; Millar Instruments, Houston, TX) were placed through the external jugular vein sheath into the right atrium and into the descending thoracic aorta through a common carotid artery sheath. Fluid-filled pigtail catheters (5F; Cordis) were advanced through the femoral vascular sheaths into the left ventricle and ascending aorta. Correct placement of all the catheters was verified by fluoroscopy. Adhesive multifunction defibrillation electrode pads (DP2/DP6; Philips Medical Systems, Seattle, WA) were placed in anteroposterior positions. A puck, containing a force transducer and an accelerometer, was adhered to the sternum with an integral adhesive pad (HeartStart4000SP, Version 0.50; Laerdal, Stavanger, Norway). Electrocardiographic leads were attached to three limbs to monitor heart rate and

rhythm. A rack (Fig. 1) was placed over the puck to hold it in position and to allow administration of compressions with no residual lean or 1.8 kg (10%) or 3.6 kg (20%) residual lean during the relaxation phase of chest compressions. The lean weight was calculated from pilot studies in similar piglets, which determined that the mean compression force required for maintaining 80–90 mm Hg peak aortic systolic pressure during CPR was 180 newtons. This force corresponds to the force of earth's gravity on an object with a mass of 18 kg. Therefore, 1.8-kg and 3.6-kg weights on the chest wall were used to represent 10% and 20% residual lean, respectively.

Measurements

Aortic and right atrial pressures, electrocardiogram, and end-tidal partial pressure of carbon dioxide were acquired by using hardware from Gould Instrument Systems (LDS, Middleton, WI), continuously displayed on a P3 Plus Ponemah Physiology Platform (DSI, St Paul, MN) and recorded on a laptop computer (Gateway, Sioux City, SD) for data analyses. All hemodynamic and mechanical data were recorded at a frequency of 200 Hz. The force transducer and accelerometer potentials generated during compressions were recorded using a modified HeartStart 4000 defibrillator with an integral feedback device (HeartStart 4000SP, Version 0.5; Laerdal). Coronary perfusion pressure was calculated as the difference between aortic pressure and right atrial pressure in the midrelaxation phase of compression. Arterial blood gases, electrolytes, and lactate analyses were periodically assessed using iStat1 (Abbott Laboratory Diagnostics, Abbott Park, IL).

The MBF and CI were determined with a neutron-activated microsphere assay technique (BioPal, Worcester, MA), as previously described (11–13). A bolus of nonradioactive microspheres (15 μm diameter) labeled with rare-earth elements (samarium, lanthanum, ytterbium, europium, lutetium, terbium, and gold suspended in iso-osmotic saline containing Tween 80 and Thimerosal) was injected into the left ventricle 30 secs after the start of each 3-min chest compression epoch. The quantity of injected microspheres was calculated using the manufacturer's recommendations for sufficient number of microspheres ($X = [1.2 \times 10^6 + 1.9 \times 105 \times \text{animal mass in kg}] \times 2$). The CI reference blood samples were obtained from the ascending aorta over 2 mins 30 secs at a rate of 10 mL/min by an automated withdrawal pump (Harvard Apparatus, South Natick, MS). An equal replacement volume of saline was immediately administered intravenously. Cardiac index for pigs was calculated using the formula published by Kelley et al (14). A 5 × 5-mm full-thickness midanterior left ventricular-free wall tissue sample was obtained postmortem and split in half for quantitative microsphere counting, which was analyzed separately as endocardial and epicardial specimens. The MBF was calculated by averaging the endocardial and epicardial blood flows and reported as $\text{mL} \cdot \text{min}^{-1} \cdot 100\ \text{g}^{-1}$ heart tissue. Quantitative microsphere measurements on blood samples and myocardial tissue samples for blood flow determinations were performed at the central laboratory of BioPal.

Experimental Protocol

After baseline data were collected, ventricular fibrillation was induced using a pacing electrode temporarily placed in the right ventricle. The presence of ventricular fibrillation was confirmed by the characteristic electrocardiographic waveform and the precipitous fall in aortic pressure. Anesthesia was discontinued and resuscitation was immediately begun using continuous manual, metronome-directed compressions at a rate of 100 compressions/min plus mechanical ventilation with room air at 10 breaths/min and a tidal volume of 15 mL/kg. The applied compression force was adjusted to attain an average aortic systolic pressure of 80–90 mm Hg during each 3-min epoch. The rescuer was blinded for all other recorded data. After 30 secs of compressions, microspheres were injected into the left ventricle as a bolus and the withdrawal pump began collecting aortic blood for 2.5 mins.

Each trial consisted of six similar 3-min epochs of chest compressions. The first and the sixth epochs were performed without any residual lean weight, whereas epochs 2–5 were randomly assigned to a sequence of either 1.8, 3.6, 1.8, and 3.6 kg of residual lean or 3.6, 1.8, 3.6, and 1.8 kg of residual lean. Defibrillation was not attempted during the experiment. A gross postmortem examination was performed to assess any unusual findings of the heart, lungs, and thorax and for obtaining tissues from the anterior free wall of the left ventricle needed for determination of MBF by an independent laboratory (BioPal, Worcester, MA).

Data Analysis

Data were entered into Microsoft Excel for Windows (Microsoft Office; Microsoft, Redmond, WA). Descriptive statistics of the raw data were calculated with SPSS version 15.0 (SPSS, Chicago, IL). Assuming that our myocardial blood flows would be 40 mL · min^{-1} · 100 g^{-1} with no lean and that the variability of measurement would be 25%, the power of this study to show a 33% decrease in myocardial blood flow with ten animals was 0.96.

Previous experiments as well as pilot experiments have shown that during long resuscitation periods (>10 mins), chest compressions are typically less effective, but the precise decrease in effectiveness of chest compressions over time is not clear (15–17). Therefore, data were modeled with five linear mixed-effect regression models using Stata Version 10.2 (StataCorp LP, College Station, TX). Time was set as a fixed effect and the pig as a random effect. The five models evaluated were as follows: 1) a linear time model; 2) three time-step models, assuming a threshold effect between epochs 2 and 3, epochs 3 and 4, and epochs 4 and 5, respectively; and 3) a log of time model. A value of $p < .05$ was considered statistically significant.

RESULTS

Ten piglets (mean weight, 10.7 ± 1.2 kg) were enrolled and studied. The mean prearrest heart rate was 115 ± 29 beats/min, the mean prearrrest systolic/diastolic aortic blood pressures were 78 ± 9/56 ± 9 mm Hg, and the mean prearrest right atrial diastolic pressure was 5 ± 1 mm Hg. Prearrest arterial blood gases revealed pH of 7.30 ± 0.04, P_{O_2} of 99 ± 9 mm Hg (13.2 ± 1.2 kPa), P_{CO_2} of 46 ± 3 mm Hg (6.1 ± 0.4 kPa), H_{CO_3} of 27 ± 2, and Sa_{O_2} of 98 ± 1%. Prearrest hemoglobin concentration was 8.0 ± 1.2 g/dL.

The mean CI and MBF at baseline during sinus rhythm and during CPR are presented in Figure 2 and Table 1. The mean CI during the first 3 mins of CPR was 1.9 ± 0.2 L · min^{-1} · M^{-2} compared with 1.1 ± 0.2 L · min^{-1} · M^{-2} during minutes 4–9 with any lean (10% or 20%; $p < .05$). The CI during minutes 10–15 with 10% or 20% lean was 0.7 ± 0.1 L · min^{-1} · M^{-2} and increased to 0.9 ± 0.2 L · min^{-1} · M^{-2} during minutes 16–18 when full chest recoil was allowed (i.e., no lean) (Fig. 2A). In addition, mean left ventricular MBF was 39 ± 7 mL · min^{-1} · 100 g^{-1} during the first 3 mins (i.e., no leaning) and decreased to 20 ± 5 mL · min^{-1} · 100 g^{-1} with 10% or 20% leaning during minutes 4–9. The MBF remained similarly low with 10% or 20% leaning during minutes 10–15 (16 ± 3 mL · min^{-1} · 100 g^{-1}) but increased substantially when full chest recoil was allowed during minutes 16–18 (25 ± 9 mL · min^{-1} · 100 g^{-1}) (Fig. 2B). As expected, the CI and MBF with no leaning were substantially lower during the last 3 mins of chest compressions compared with the first 3 mins (0.9 ± 0.2 L · min^{-1} · M^{-2} vs. 1.9 ± 0.3 L · min^{-1} · M^{-2} and 25 ± 9 mL · min^{-1} · 100 g^{-1} vs. 38 ± 11 mL · min^{-1} · 100 g^{-1}).

The logarithm of time model was the best fit among the five evaluated models (18). Adjusted for the log of time, the CI was significantly reduced from 1.9 ± 0.2 to 1.6 ± 0.1 L · M^{-2} · min^{-1} for 10% leaning and was further reduced to 1.4 ± 0.2 L · M^{-2} · min^{-1} for 20%

leaning ($p < .05$). The MBF with no lean was 39 ± 7 mL · min^{-1} · $100g^{-1}$ and decreased to 30 ± 6 mL · min^{-1} · $100g^{-1}$ with 10% leaning and 26 ± 6 mL · min^{-1} · $100g^{-1}$with 20% leaning ($p < .05$) each compared with no lean. Interestingly, the CI and MBF with 10% lean were not statistically different from CI and MBF with 20% lean. As planned, the adjusted aortic systolic pressures were not different among any of the six chest compression epochs. The mean right atrial diastolic pressure increased with leaning (9 ± 0.6 mm Hg with no lean, 10 ± 0.3 mm Hg with 10% lean, and 13 ± 0.3 mm Hg with 20% lean; $p < .01$) and the mean coronary perfusion pressure decreased (Fig. 3).

DISCUSSION

This study establishes that residual leaning of only 1.8 kg and 3.6 kg (approximately 10% and 20% of the force of the chest compressions) can substantially decrease CI and MBF during CPR. Surprisingly, the hemodynamic effects of 10% and 20% residual lean were similar, suggesting that the hemodynamic effects of leaning were primarily the result of the lack of full chest recoil. Furthermore, this study demonstrates that CI and MBF progressively decrease over time during prolonged CPR. Nevertheless, removal of leaning appeared to improve hemodynamics even after 16 mins of CPR.

The effects of leaning were demonstrable throughout 18 mins of CPR both during the first 9 mins (early) and the last 9 mins (later) (Fig. 2; Table 1). For example, during the first 9 mins of CPR, the right atrial end-diastolic pressure increased from 9 ± 1 mm Hg with no lean to 11 ± 1 mm Hg and 13 ± 1 mm Hg with 10% and 20% lean, respectively, presumably because of transthoracic transmission of the leaning weight to the venous system. These higher venous pressures resulted in lower coronary perfusion pressure and the concomitant substantial decreases in MBF from 39 ± 7 mL · 100 g^{-1} · min^{-1} with no lean to 30 ± 10 mm Hg with 10% lean and 26 ± 10 mm Hg with 20% lean. In addition, the CI decreased from 1.9 ± 0.3 L · min^{-1} · M^{-2} with no lean to 1.2 ± 0.2 L · min^{-1} · M^{-2} with 10% lean and 1.0 ± 0.1 L · min^{-1} · M^{-2} with 20% lean. Interestingly, these seemingly modest changes in vascular pressures were associated with substantial changes in blood flows during the low-flow hemodynamic state of CPR. Although both 10% and 20% lean resulted in significantly different hemodynamics during CPR compared with no lean, they were not demonstrably different from one another. Both 10% and 20% leaning prevented full chest recoil and presumably impaired the generation of negative intrathoracic pressure, thereby limiting venous return and cardiac output. In this study, inhibiting the generation of negative intrathoracic pressure that would have been attained with full chest recoil was apparently a more important factor than the relative amount of leaning. Importantly, the adverse hemodynamic effects of 10% and 20% leaning remained significant after controlling for the expected hemodynamic deterioration during prolonged CPR (Fig. 3).

The American Heart Association Guidelines for Emergency Cardiovascular Care and Cardiopulmonary Resuscitation recommend target values for selected CPR parameters to optimize the effectiveness of CPR (1, 2). In the 2005 Guidelines, complete release of sternal pressure between chest compressions is specifically recommended with the intent to maximize venous return by allowing full chest recoil (1, 2). The 2005 Guidelines highlight data from Aufderheide et al (3) showing that residual and continuous pressure on the chest wall was demonstrable during the decompression phase of CPR at some time in six of 13 patients during emergency medical services-provided out-of-hospital resuscitative efforts. In other data collected before 2005, Tomlinson et al (19) documented that rescuers frequently do not allow full chest recoil. They showed that the average residual force, or “lean,” during adult out-of-hospital CPR was 1.7 ± 1.0 kg, corresponding to an average residual depth of 3 ± 2 mm. Interestingly, that average residual lean was quite similar to the 1.8-kg lean in our piglet investigation that corresponded to 10% of the force required to maintain 85 mm Hg

aortic compression pressures. Recently, Niles et al (4) showed that residual leaning of 2.5 kg was demonstrable in 50% of inhospital chest compressions for children 8 to 18 yrs old when automated feedback was not provided.

Yannopoulos et al (20) have demonstrated that incomplete chest wall recoil during the decompression phase of CPR in adult pigs can increase endotracheal and right atrial pressure as well as decrease systolic, diastolic, and mean aortic pressures and coronary and cerebral perfusion pressures. In their model, CPR without “lean” was provided by a pneumatically driven automatic piston device compressing the chest wall 25% of the anteroposterior diameter. Residual lean was added by reducing the decompression distance to 75% of its full excursion in the decompression phase. These authors acknowledged that their definition of incomplete chest wall decompression (i.e., residual “leaning”) was an important limitation of their study because the decreases in hemodynamics may have resulted from the 25% decreased stroke length with their chest compressions and concomitant decrease in force of compressions rather than the residual lean *per se*. We therefore chose to target the compression effort to attain aortic systolic pressures of 80–90 mm Hg during CPR rather than specifically limiting the force or depth of compressions. Furthermore, they reported the effect of reduced decompression distance only on hemodynamic pressures and not on blood flows. Our swine investigation is the first to specifically address the effects of residual leaning force on CPR hemodynamics without the confounding issue of *a priori* prescriptive decreases in the amount of compression depth/force. More importantly, our investigation is the first to demonstrate that residual chest wall leaning decreases myocardial and systemic blood flows during CPR.

Novel methods and technology to monitor CPR quality have been recently developed that allow the quantification of the thoracic response to chest compressions. Specifically, a load cell and accelerometer sensor package has been integrated into a clinical monitor/defibrillator to track chest compression and applied force during CPR (21–23). The sensor is interposed between the palms of the hands of the person administering CPR and the sternum of the patient. The accelerometer signal is processed with a double-integration algorithm, yielding deflection. These devices can measure and provide feedback on the rate, depth, force, and sternal pressure release during CPR. This automated directive and corrective feedback can improve the quality of CPR delivered during training and during actual CPR delivery (6, 8). Thus, the technology exists to provide feedback to providers when they are “leaning” on the chest during CPR.

There were several limitations with this model. This animal study used healthy young piglets, and the quality of CPR was excellent. The effects of residual leaning during CPR in sick children or when poor-quality CPR is administered may yield different results. Additional studies in humans are needed to evaluate the effects of residual leaning force on intrathoracic pressure, vascular pressures, venous return to the heart, and coronary and cerebral perfusion. In this experiment, hemodynamic parameters changed over time, consistent with previously well-documented changes in vascular pressures and blood flows with prolonged CPR (15–17). Therefore, we accounted for this potentially confounding factor *a priori* with a linear mixed-effect regression model. We evaluated five such models adjusting this time effect, and the model with the best fit was the log of time model used. This log model operates like an exponential decay (i.e., performance declines over time but at a decreasing rate).

CONCLUSIONS

In this piglet model of CPR for ventricular fibrillation, modest residual leaning of 10% (1.8 kg) and 20% (3.6 kg) of the force required to attain aortic systolic pressure at 85 mm Hg had

substantial effects on CI and MBF during CPR. Because such leaning is common during actual clinical CPR performance, these findings have important implications with regard to quality and effectiveness of CPR. These observations underline the importance of developing directive and corrective CPR feedback systems to minimize leaning during pediatric CPR.

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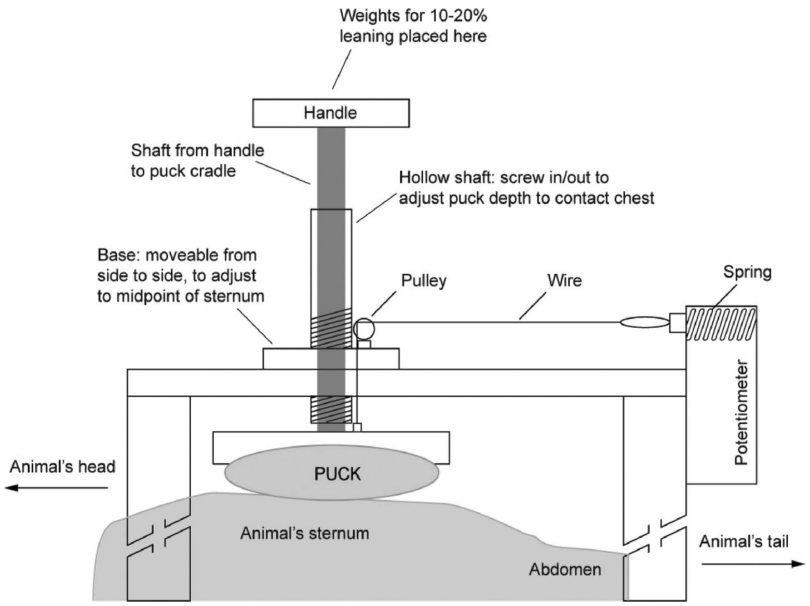

Figure 1.
Graphic display of piglets and cardiopulmonary resuscitation equipment.

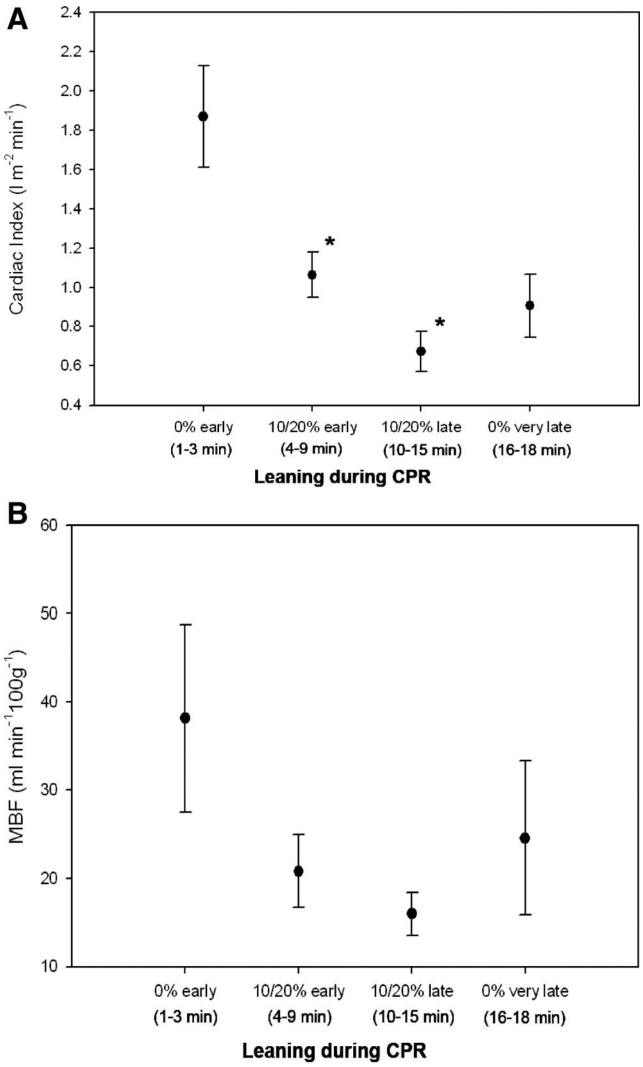

Figure 2.

A–B, Time-dependent effects of leaning on blood flows during CPR. Shown are the cardiac index data (*A*) and the left ventricular myocardial blood flow data (*B*). Black dots are the means, and error bars refer to SEM. "10%/20% early" refers to the mean combined blood flows with 10% and 20% lean (1.8 kg and 3.6 kg, respectively) from 4 to 9 mins of CPR. "10%/20% late" refers to the mean combined blood flows with 10% and 20% lean (1.8 kg and 3.6 kg, respectively) from 10 to 15 mins of CPR. MBF refers to left ventricular myocardial blood flow. *Differs from no lean (i.e., "0% early") by Mann-Whitney *U* test, $p < .05$. Using the linear mixed-effects regression model to account for CPR changes over time, CI and MBF each decreased after 10% and 20% lean compared with no lean (including "0% early" and "0% very late"); $p < .05$. *CPR*, cardiopulmonary resuscitation; *MBF*, myocardial blood flow; *CI*, cardiac index.

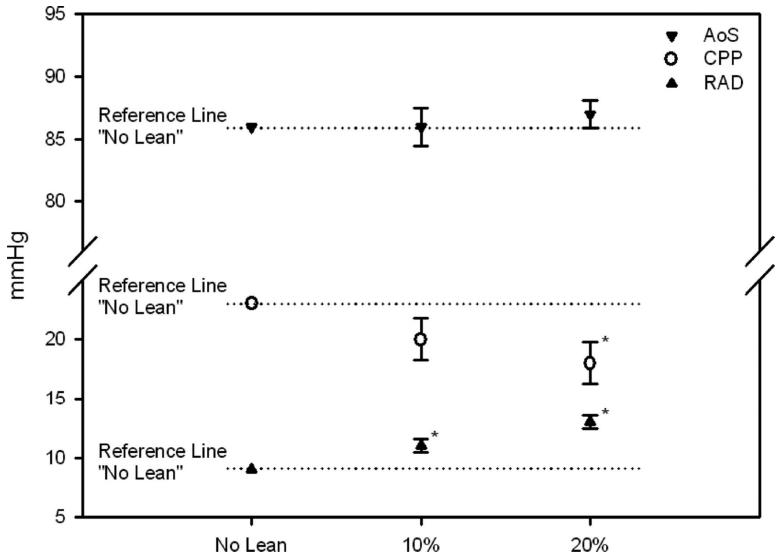

Figure 3.

Effects of 10% and 20% leaning on hemodynamic pressures during cardiopulmonary resuscitation adjusted for log of time. Values are mean ± SEM. *Differs from no lean (reference line) by linear missed-effects regression model ($p < .05$). *CPP*, coronary perfusion pressure; *RAD*, right atrial diastolic pressure; *AoS*, aortic systolic pressure.

Table 1

Blood flow data

Time, mins	Lean	Cardiac Index, L · M^{-2} · min^{-1} Mean ± sem (n)	Left Ventricular Myocardial Blood Flow, mL · min^{-1} · 100 g^{-1} Mean ± sem (n)
Baseline (sinus rhythm)	4.5 ± 0.4 (9)	101 ± 16 (9)	
During resuscitation			
1–3	No lean	1.9 ± 0.3 (9)	38 ± 11 (9)
4–9	10%	1.2 ± 0.2 (9)	23 ± 7 (9)
4–9	20%	1.0 ± 0.1 (8)	19 ± 4 (8)
10–15	10%	0.7 ± 0.2 (9)	17 ± 4 (9)
10–15	20%	0.7 ± 0.1 (8)	15 ± 3 (8)
16–18	No lean	0.9 ± 0.2 (9)	25 ± 9 (9)

“10%” and “20%” lean refer to 1.8 kg and 3.6 kg residual weight, respectively, on the chest wall during the relaxation phase of cardiopulmonary resuscitation.