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Prevalence and hemodynamic effects of leaning during CPR

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Abstract

Background—Cardiopulmonary resuscitation (CPR) guidelines recommend complete release between chest compressions (CC).

Objective—Evaluate the hemodynamic effects of leaning (incomplete chest wall release) during CPR and the prevalence of leaning during CPR.

Results—In piglet ventricular fibrillation cardiac arrests, 10% and 20% (1.8 kg and 3.6 kg, respectively), leaning during CPR increased right atrial pressures, decreased coronary perfusion pressures, and decreased cardiac index and left ventricular myocardial blood flow by nearly 50%. In contrast, residual leaning of a 260 g accelerometer/ force feedback device did not adversely affect cardiac index or myocardial blood flow. Among 108 adult in-hospital CPR events, leaning 2.5 kg was demonstrable in 91% of the events and 12% of the evaluated CC. For 12 children with in-hospital CPR, 28% of CC had residual leaning 2.5 kg and 89% had residual leaning 0.5 kg.

Conclusions—Leaning during CPR increases intrathoracic pressure, decreases coronary perfusion pressure, and decreases cardiac output and myocardial blood flow. Leaning is common during CPR.

Keywords

Cardiac arrest; Cardiopulmonary resuscitation; CPR; Children; Pediatric; Leaning; Hemodynamics; Intrathoracic pressure; Incomplete release; Force transducer; Chest recoil

Introduction

Quality of cardiopulmonary resuscitation (CPR) is critical for survival and good neurological outcome following cardiac arrest.^{1,2} CPR guidelines recommend target values for rate and depth of chest compressions and ventilations, limiting interruptions in chest compressions, and allowing complete release (full chest recoil) between chest compressions.³ Interestingly, the 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care added the recommendation to “allow full chest recoil” despite limited data on its hemodynamic importance or the prevalence of incomplete release (leaning).⁴

1.1. Hemodynamic effects of leaning

An early suggestion that incomplete chest wall release should be discouraged during CPR was provided in the 1961 landmark work of Jude, Kouwenhoven, and Knickerbocker: “Cardiac venous filling is considered to be enhanced by the maintenance intact of the pleural integrity with external massage. With relaxation following each external compression, the sternum recoils outward increasing intrathoracic negative pressure and thereby aiding in the venous filling phase of the heart. This point is considered to be of great importance in providing a good cardiac output in the arrested heart.”⁵

1.2. Porcine models in cardiac arrest

Yannopoulos and colleagues evaluated the effects of incomplete chest wall release in nine pigs with automated CPR during 6 min of ventricular fibrillation (VF).⁶ A CC depth of 25% of the anterior–posterior diameter with 100% decompression (standard CPR) was provided for 3 min. CC was then reduced to 75% of complete decompression for 1 min of CPR (so that the device did not “release” the last 25% of the stroke length) and then restored for another 1 min of CPR to 100% full decompression. In these pigs, incomplete release (equaling approximately 12 mm) resulted in significant increases in endotracheal pressure and right atrial pressure, and significant decreases in systolic, diastolic, mean arterial, coronary and cerebral perfusion pressures (Table 1).

The combination of the increase of decompression phase right atrial pressure and the decrease of aortic arterial pressure, significantly and immediately decreased coronary perfusion pressure (CPP) to 15.1 ± 1.6 mm Hg. However, the experimental protocol could not differentiate whether the decrease in CPP was due to a decrease by 25% in the compression stroke length or the lack of full chest recoil.

Zuercher et al. therefore evaluated the effects of leaning while continuing to provide a consistent aortic compression pressure during CPR for 10 piglets in ventricular fibrillation.⁷ First, they determined that the mean compression force required to maintain 80–90 mm Hg peak aortic systolic pressure during CPR was 180 newtons, thereby requiring a mean weight of 18 kg. They, therefore, defined 10% and 20% residual leaning as 1.8 kg and 3.6 kg residual leaning weight on the chest wall, respectively. CCs were provided manually to maintain average aortic systolic pressure of 80–90 mm Hg during each of six 3 min CPR epochs (total CPR duration of 18 min). The first and the 6 epochs were performed without any residual lean weight, whereas epochs 2–5 were randomly assigned to a sequence of 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. In these piglets, a residual leaning force of 1.8–3.6 kg during CPR substantially increased right atrial diastolic pressure and decreased CPP (Table 2). Most importantly, this apparently “minor” leaning of only 1.8 kg decreased cardiac index and myocardial blood flow by nearly 50% (Fig. 1).

Why is the adverse effect of 10% and 20% lean during CPR so similar? The authors presumed that both quantities of leaning impaired full elastic recoil of chest wall and that the last bit of recoil may be very important to create adequate negative intrathoracic pressure.

To further address this issue, Zuercher and colleagues evaluated the effects of even smaller leaning force/weight during CPR.⁸ They evaluated 260 g of residual leaning, equal to a commercially available sternal accelerometer/force feedback sensor device. The investigative team studied the effect of such leaning among piglets during 12 min of chest compressions during ventricular fibrillation (VF) cardiac arrest. Four 3-min CPR epochs were randomized to CCs with the sensor (260 g leaning force) or without the sensor (no leaning force). Interestingly, the use of a 260 g accelerometer/force feedback sensor did not adversely affect cardiac output or left ventricular myocardial blood flow in these piglets during CPR. These data suggest that such a small leaning weight did not prevent full recoil of the chest wall with its concomitant decrease in intrathoracic pressure.

The minimal threshold at which leaning has deleterious effects on hemodynamics may be most important when we consider the pediatric population because the chest wall is more compliant and may allow greater intrathoracic transmission of residual leaning through the chest wall. Two studies have looked at real pediatric patients in order to determine the minimal leaning forces that would affect intrathoracic and CPP.

Sutton et al. investigated 13 healthy, anesthetized, paralyzed mechanically ventilated children (age: 26 ± 24 m, range: 6.5–87 m; weight: 13 ± 5 kg, range: 7.4–24.8 kg).⁹ Sternal force (leaning) was defined as 10% to 25% of the subject's body weight. Peak endotracheal pressure (ETP), a surrogate of intrathoracic pressure (ITP), was continuously measured before and during serial incremental increases in sternal force from 10% to 25%. A delta ETP of 2.0 cm H₂O was considered clinically significant.

In these healthy anesthetized children, changes in ITP (as determined by ETP) were significant at residual leaning forces as low as 10% of subject body weight (Fig. 2). Residual leaning force of 2.5 kg was also significantly associated with increases in ETP 2.0 cm H₂O. The authors recognized that the main limitation of this investigation was that the subjects were healthy and had spontaneous circulation during ETP monitoring. Therefore, while modest changes in ETP were demonstrated, it is possible that the model used in their investigation differs from the arrested child in both chest wall compliance and thoracic muscular tone. Additionally, the effects of ETP changes on blood flow were not measured.

In order to better determine the minimal leaning forces that would affect central venous hemodynamics, Glatz et al. studied 14 anesthetized children during cardiac catheterization (mean age: 5.4 ± 1.6 yrs, range: 3.1–7.9 yrs; weight: 18.5 ± 3.5 kg).¹⁰ Sternal force (leaning) was defined as 10% to 25% of the subject's body weight. Central hemodynamic measures via right heart and arterial catheters and intrathoracic pressure (ITP) (as determined by ETP) via pneumotachometer were measured before and during incremental increases in leaning force of 10 and 20% of body weight. In these healthy, anesthetized children, mean right atrial pressure increased significantly with leaning force as low as 10% of subject body weight, and increased even more at 20%. ITP also increased significantly with leaning force of 10%, and even more with 20%. Importantly, CPP (aortic diastolic – mRAP) decreased significantly with leaning forces of 10% and 20%. However, no significant differences were detected for other hemodynamic measures in these children with normal hemodynamics. In contrast, such increases in intrathoracic pressure may well impede venous return in the low flow state of CPR.

These laboratory investigations have demonstrated that incomplete release during chest compressions (using the various definitions of leaning force) can have significant deleterious

effects on hemodynamics. Leaning during chest compressions significantly increases in endotracheal pressure and right atrial pressure, and significantly decreases systolic, diastolic, mean arterial pressures and coronary and cerebral perfusion pressures.

2. Prevalence of leaning during clinical CPR and simulation CPR

2.1. Adults

Various clinical adult studies have suggested that leaning is common in out-of-hospital (OOH) and in-hospital (IH) cardiac arrest resuscitations. Aufderheide and colleagues, using a definition of leaning in adults as >2 mm depth, reported that Emergency Medical Services (EMS) BLS-certified rescuers maintained some residual leaning at some time during resuscitative efforts in 46% (6/13) of adult OOH cardiac arrest victims.¹¹ They found that during those events, airway pressures were consistently positive during the decompression phase (>0 mm Hg) which may be attributed to a number of factors (incomplete release of the chest, prolonged positive ventilations, intrinsic positive end expiratory pressure, increased airway resistance, or some combination of the above).

A study of IH cardiac arrest resuscitations by Fried et al. analyzed 112,569 chest compressions from 108 adult CPR events and found that chest compression with leaning forces ≥ 2.5 kg occurred in 98/108 (91%) of those events.¹² There was a wide distribution of leaning among the events and chest compression blocks (30 s of CC without pauses). Leaning affected as many as 44% of chest compressions in a given event. Although >2.5 kg of leaning was observed in >90% of the CPR events, this amount of leaning was demonstrable in only 112,569/13,270 (12%) of all chest compressions.

2.2. Pediatrics

The first study to determine prevalence of leaning in real pediatric cardiac arrests, Niles et al. defined complete release as a leaning force less than 0.5 kg.¹³ Our study analyzed 37,085 CCs from 20 IH cardiac arrest resuscitations. For all CCs delivered, more than 89% had residual leaning force ≥ 0.5 kg and over 28% had a leaning force ≥ 2.5 kg. Additionally, it was determined that the median leaning force was 1.6 kg (IQR 0.9–2.7 kg) with a leaning depth of 2.9 (1.6–4.7) mm.

2.3. Manikins

In a study of simulated cardiac arrest in a controlled setting using a recording manikin, Aufderheide and colleagues observed the prevalence of incomplete chest wall decompression (release) by emergency medical services (EMS) personnel during CPR.¹¹ Incomplete release was defined as any release attempt that did not return to within 2 mm of baseline (the limit of measurement of the recording manikin). Of 6,202 CCs with standard hand positioning, only 16.3% of all compression–decompression cycles exhibited complete chest wall release. The authors noted that rescuer fatigue appeared to contribute to incomplete chest wall release (>2.5 kg of residual leaning) in their clinical observation study, but that the nearly universal occurrence of leaning in this more sensitive set of manikin measurements suggests that leaning is a much more common problem and occurs even without rescuer fatigue.

3. Conclusions

Both pig and human studies demonstrate that leaning can have substantial negative hemodynamic consequences on cardiac output and coronary and cerebral perfusion. During both simulated and real human cardiac arrest studies, the occurrence of leaning is common.

As surmised by Jude, Kouwenhoven and Knickerbocker in 1961, attention to minimize leaning during CPR can improve CPR quality and efficacy.

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Conflict of interest

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Fig. 1.
Graph of aortic systolic (AoS), right atrial diastolic (RAD), coronary perfusion pressure (CPP) changes at baseline (no lean) and with residual lean of 10% and 20% (mm Hg).⁷

Fig. 2.
Sample subject recording. Peak endotracheal pressure (ETP) vs. time (s).⁹

Fig. 3. Mean right atrial pressure (mm Hg) changes with application of sternal weights (lean) at baseline (no lean) and 10% and 20% of body weight.¹⁰

Table 1

Aortic systolic (AoS), right atrial diastolic (RAD) and coronary perfusion pressure (CPP) measurements (mm Hg) without and with 25% leaning.¹

	Std CC	CC w/ Lean
AoS	76 ±4	65 ±5
RAD	4.8 ±1.3	5.6 ±1.2
CPP	23.3 ±1.9	15.1 ±1.6*

Table 2

Changes in aortic systolic (AoS), right atrial diastolic (RAD), coronary perfusion pressure (CPP), myocardial blood flow (MBF) and cardiac index (CI) at baseline (no lean) and with residual lean of 10% and 20% (mm Hg).⁷

	No lean	10%	20%
AoS	87 ±4	86 ±4	87 ±5
RAD	9 ±2	11 ±2 *	13 ±3 *
CPP	22 ±5	19 ±7 [#]	17 ±6 [#]
MBF	0.40 ±0.34 [†]	0.24 ±0.22	0.19 ±0.12
CI	1.78 ±0.77 [†]	1.21 ±0.61	0.95 ±0.32

* $P < 0.001$

[#] $P < 0.05$.

[†] $P < 0.05$, No lean vs. 10% & 20%.