ZOLL®
Advancing Resuscitation. Today.™

AutoPulse®
Non-invasive Cardiac Support Pump

Resumo: Performance hemodinâmica melhorada com um novo dispositivo de compressão torácica durante tratamento em parada cardíaca dentro de hospitais

Sergio Timerman[a], Luis Francisco Cardoso[a], Jose A.F. Ramires[a], Henry Halperin[b]

a- The Heart Institute (InCor), Sao Paulo University School of Medicine, Sao Paulo, Brazil.
b- Cardiology Division, Departments of Medicine, Biomedical Engineering, and Radiology, Johns Hopkins Medical Institutions, Blalock 524, 600 N. Wolfe Street, Baltimore, MD 21205, USA

Introdução

Introdução: O objectivo deste estudo clínico piloto foi determinar se um novo dispositivo de compressão torácica poderia melhorar a hemodinâmica quando comparado com a compressão torácica manual durante ressuscitação cardio-pulmonar (RCP) em humanos. O dispositivo é um compressor torácico electromecânico automático ajustável baseado na tecnologia AutoPulse™ (Zol Medical Corpotation) que utiliza uma banda de compressão distribuidora de carga (RCP-A) para comprimir a parede torácica anterior.

Métodos

Foram avaliados, com aprovação do corpo de revisores da instituição, um total de 31 doentes sequenciais com parada cardíaca súbita intra-hospitalar. Todos os doentes receberam tratamento prévio para doença cardíaca e a maioria tinha co-morbilidades. Os doentes foram incluídos após 10 min de protocolo de suporte avançado de vida (SAV) standard sem sucesso. Foram introduzidos cateteres preenchidos com fluído na aorta torácica e na aurícula direita e confirmada a sua localização pelas ondas de pressão e radiografia torácica. A pressão de perfusão coronária (PPC) foi medida como a diferença entre as pressões aórtica e auricular direita durante a fase de descompressão torácicas. Após 10 minutos de SAV sem sucesso e colocação dos cateteres, os doentes receberam compressões torácicas manuais e RCP-A alternadas durante 90s cada. As compressões torácicas foram administradas sem pausa para ventilação a 100 compressões/minuto e 60 compressões/minuto, respectivamente para RCP manual e RCP-A. Todos os doentes receberam intubação endotraqueal e foram ventilados por máscara com insuflador a 12 ventilações/min entre as compressões. Foi dada adrenalina (1mg bólus iv) conforme solicitado pelo médico responsável a intervalos de 3–5 min. Estiveram presentes registos de pressões utilizáveis em 16 doentes (68 ± 6 anos, 5 mulheres), e só se apresentam os dados desses doentes. As compressões torácicas RCP-A aumentaram a pressão aórtica máxima em comparação com as compressões torácicas manuais (153 ± 28mmHg contra 115 ± 42 mmHg, $P < 0.0001$, média ± S.D.). Da mesma forma a RCP-A aumentou a pressão máxima na aurícula direita em relação à compressão torácica manual (20 ± 12mmHg contra 15 ± 11 mmHg, $P < 0.015$). As compressões torácicas foram de elevada qualidade, consistente (51±20 Kg) e emtodos os casos atingiram ou excederam as recomendações da American Heart Association para a profundidade das compressões.

Conclusão

Em investigação prévia demonstrou-se que o aumento da PPC se correlaciona com maior fluxo sanguíneo coronário e melhores taxas de retorno de circulação na paragem cardíaca súbita. O sistema RCP-A utilizando tecnologia AutoPulse demonstrou ser capaz de aumentar a pressão de perfusão coronária quando comparado com compressão torácica manual durante RCP nesta população de doentes graves.

Na próximas páginas segue cópia do artigo original.

ELSEVIER

Resuscitation 61 (2004) 273–280

RESUSCITATION

www.elsevier.com/locate/resuscitation

Improved hemodynamic performance with a novel chest compression device during treatment of in-hospital cardiac arrest

Sergio Timerman [a], Luis Francisco Cardoso [a], Jose A.F. Ramires [a], Henry Halperin [b,*]

[a] *The Heart Institute (InCor), Sao Paulo University School of Medicine, Sao Paulo, Brazil*
[b] *Cardiology Division, Departments of Medicine, Biomedical Engineering, and Radiology, Johns Hopkins Medical Institutions, Blalock 524, 600 N. Wolfe Street, Baltimore, MD 21205, USA*

Received 15 June 2003; received in revised form 8 January 2004; accepted 8 January 2004

Abstract

Introduction: The purpose of this pilot clinical study was to determine if a novel chest compression device would improve hemodynamics when compared to manual chest compression during cardiopulmonary resuscitation (CPR) in humans. The device is an automated self-adjusting electromechanical chest compressor based on AutoPulse™ technology (Revivant Corporation) that uses a load distributing compression band (A-CPR) to compress the anterior chest. *Methods*: A total of 31 sequential subjects with in-hospital sudden cardiac arrest were screened with institutional review board approval. All subjects had received prior treatment for cardiac disease and most had co-morbidities. Subjects were included following 10 min of failed standard advanced life support (ALS) protocol. Fluid-filled catheters were advanced into the thoracic aorta and the right atrium and placement was confirmed by pressure waveforms and chest radiograph. The coronary perfusion pressure (CPP) was measured as the difference between the aortic and right atrial pressure during the chest compression's decompressed state. Following 10 min of failed ALS and catheter placement, subjects received alternating manual and A-CPR chest compressions for 90 s each. Chest compressions were administered without ventilation pauses at 100 compressions/min for manual CPR and 60 compressions/min for A-CPR. All subjects were intubated and ventilated by bag-valve at 12 breaths/min between compressions. Epinephrine (adrenaline) (1 mg i.v. bolus) was given at the request of the attending physician at 3–5 min intervals. Usable pressure signals were present in 16 patients (68 ± 6 years, 5 female), and data are reported from those patients only. A-CPR chest compressions increased peak aortic pressure when compared to manual chest compression (153 ± 28 mmHg versus 115 ± 42 mmHg, $P < 0.0001$, mean ± S.D.). Similarly, A-CPR increased peak right atrial pressure when compared to manual chest compression (129 ± 32 mmHg versus 83 ± 40 mmHg, $P < 0.0001$). Furthermore, A-CPR increased CPP over manual chest compression (20 ± 12 mmHg versus 15 ± 11 mmHg, $P < 0.015$). Manual chest compressions were of consistent high quality (51 ± 20 kg) and in all cases met or exceeded American Heart Association guidelines for depth of compression. *Conclusion*: Previous research has shown that increased CPP is correlated to increased coronary blood flow and increased rates of restored native circulation from sudden cardiac arrest. The A-CPR system using AutoPulse technology demonstrated increased coronary perfusion pressure over manual chest compression during CPR in this terminally ill patient population.

Keywords: Cardiopulmonary resuscitation; Coronary perfusion pressure; Cardiac arrest; Chest compression

Resumo

Introdução: O objectivo deste estudo clínico piloto foi determinar se um novo dispositivo de compressão torácica poderia melhorar a hemodinâmica quando comparado com a compressão torácica manual durante reanimação cardio-pulmonar (CPR) em humanos. O dispositivo é um compressor torácico electromecânico automático ajustável baseado na tecnologia AutoPulse™ (Revivant Corporation) que utiliza uma banda de compressão distribuidora de pressão (A-CPR) para comprimir a parede torácica anterior. *Métodos*: Foram avaliados, com aprovação do corpo de revisores da instituição, um total de 31 doentes sequenciais com paragem cardíaca súbita intra-hospitalar. Todos os doentes receberam tratamento prévio para doença cardíaca e a maioria tinha co-morbilidades. Os doentes foram incluídos após 10 min de protocolo de suporte avançado de vida (ALS) standard sem sucesso. Foram introduzidos cateteres preenchidos com fluído na aorta torácica e na aurícula direita e confirmada a sua localização pelas ondas de pressão e radiografia torácica. A pressão de perfusão coronária (CPP) foi medida como a diferença entre as pressões aórtica e auricular direita durante a fase de descompressão torácicas. Após 10 minutos de ALS sem sucesso

* Corresponding author.
E-mail address: hhalper@jhmi.edu (H. Halperin).

0300-9572/$ – see front matter © 2004 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.resuscitation.2004.01.025

e colocação dos cateteres, os doentes receberam compressões torácicas manuais e A-CPR alternadas durante 90s cada. As compressões torácicas foram administradas sem pausa para ventilação a 100 compressões/minuto e 60 compressões/minuto, respectivamente para CPR manual e A-CPR. Todos os doentes receberam intubação endotraqueal e foram ventilados por máscara com insuflador a 12 ventilações/min entre as compressões. Foi dada adrenalina (1mg bólus iv) conforme solicitado pelo médico responsável a intervalos de 3–5 min. Estiveram presentes registos de pressões utilizáveis em 16 doentes (68 ± 6 anos, 5 mulheres), e só se apresentam os dados desses doentes. As compressões torácicas A-CPR aumentaram a pressão aórtica máxima em comparação com as compressões torácicas manuais (153 ± 28 mmHg versus 115 ± 42 mmHg, $P < 0.0001$, média ± S.D.). Da mesma forma a A-CPR aumentou a pressão máxima na aurícula direita em relação à compressão torácica manual (20 ± 12 mmHg versus 15 ± 11 mmHg, $P < 0.015$). As compressões torácicas foram de elevada qualidade, consistente (51 ± 20 Kg) e em todos os casos atingiram ou excederam as recomendações da American Heart Association para a profundidade das compressões. *Conclusão*: Em investigação prévia demonstrou-se que o aumento da CPP se correlaciona com maior fluxo sanguíneo coronário e melhores taxas de retorno de circulação na paragem cardíaca súbita. O sistema A-CPR utilizando tecnologia AutoPulse demonstrou ser capaz de aumentar a pressão de perfusão coronária quando comparado com compressão torácica manual durante CPR nesta população de doentes graves.

Palavras chave: Reanimação cardio-pulmonar; Pressão de Perfusão Coronária; Paragem cardíaca; Compressão torácica

Resumen

Introducción: El propósito de este estudio clínico piloto fue determinar si un nuevo dispositivo para compresiones torácicas podría mejorar la hemodinamia si se compara con compresiones torácicas manuales durante la reanimación cardiopulmonar (CPR) en humanos. El dispositivo es un compresor torácico electromecánico auto ajustable basado en tecnología AutoPulse™ (Revivant Corporation) que utiliza una banda de distribución de carga de compresión (A-CPR) para comprimir el tórax anterior. *Métodos*: Se estudiaron un total de 31 pacientes secuenciales con paro cardíaco súbito intra hospitalario con la aprobación de un comité de revisión institucional. Todos los sujetos recibieron tratamientos previos para enfermedad cardíaca y la mayoría tenía comorbilidades. Los sujetos fueron incluidos después de 10 minutos de protocolo de soporte vital avanzado (ALS) estándar . Se avanzaron catéteres llenados con fluidos hasta la aorta torácica y hasta la aurícula derecha, y su localización fue confirmada por ondas de presión y radiografías de tórax. La presión de perfusión coronaria (CPP) fue medida como la diferencia entre la presión aórtica y la de aurícula derecha durante la fase de descompresión de la compresión torácica. Después de 10 minutos de ALS no exitoso y de la ubicación de los catéteres, los sujetos recibieron alternadamente compresiones torácicas manuales y A-CPR por 90s cada uno. Las compresiones fueron administradas sin pausas de ventilación a 100 compresiones/min para CPR manual y 60 compresiones/min por A-CPR. Todos los sujetos recibieron intubación endotraqueal y fueron ventilados con bolsa máscara a 12 ventilaciones por minuto entre compresiones. Se aplicó Epinefrina (bolo de 1mg i.v.) a la solicitud del médico tratante a intervalos de 3–5 min. Se encontraron signos de presión utilizables en 16 pacientes (68 ± 6 años, 5 mujeres), y los datos son reportados solo para esos pacientes. Las compresiones con A-CPR aumentaron la presión máxima aórtica cuando se compara con compresiones torácicas manuales (153 ± 28 mmHg versus 115 ± 42 mmHg, $P < 0.0001$, promedio ± D.S.). Similarmente, la A-CPR aumentó la presión máxima en aurícula derecha cuando se la compara con compresiones torácicas manuales (129 ± 32 mm Hg versus 83 ± 40 mm Hg, $P < 0.0001$). Mas aun, la A-CPR aumentó la CPP sobre las compresiones torácicas manuales (20 ± 12 mmHg versus 15 ± 11 mmHg, $P < 0.015$). Las compresiones manuales fueron de calidad (51 ± 20 kg) y en todos los casos alcanzaban o superaban las guías de la AHA para profundidad de compresiones. *Conclusiones*: La investigación previa ha mostrado que CPP aumentada está correlacionada con flujo coronario elevado y tasas elevadas de retorno a circulación después de un paro cardíaco. El sistema A-CPR usando la tecnología AutoPulse demostró CPP elevada por encima de las presiones obtenidas con compresiones manuales durante CPR manual en esta población con enfermedades terminales.

Palabras clave: Reanimación cardiopulmonar; Presión de perfusión coronaria; Paro cardíaco; Compresión torácica.

1. Introduction

There are over 400,000 victims of cardiac arrest each year in the United States, and resuscitation attempts are generally unsuccessful [1]. When defibrillation fails or is not indicated, both laboratory studies [2,3] and clinical studies [4] have shown that restoration of cardiac function after cardiac arrest is related to the level of vascular pressures generated during resuscitation, especially the coronary perfusion pressure. Coronary perfusion pressures above 15 mmHg are typically needed for successful resuscitation [4].

More recent clinical reports have shown that circulation can improve the success of resuscitation even when defibrillation is indicated. Manual CPR was applied to arrest victims with out-of-hospital ventricular fibrillation (VF) for 90 s prior to defibrillation attempts resulting in an improvement in survival from 24 to 30% [5]. A later randomized clinical trial evaluated a similar group of patients with 3 min of manual CPR prior to defibrillation and demonstrated an improvement in survival from 15 to 22% [6]. These studies show that circulation can be an effective component of resuscitation even in cardiac arrest victims with VF. In patients with rhythms of asystole or pulseless electrical activity, CPR is the only treatment available.

The overall objective of the present study was to evaluate the ability of a novel non-invasive chest compression device using AutoPulse™ technology (Revivant Corporation, Sunnyvale, CA) to generate circulation (A-CPR). A-CPR is based on the concept that distributing force over the anterior chest improves the effectiveness of chest compressions.

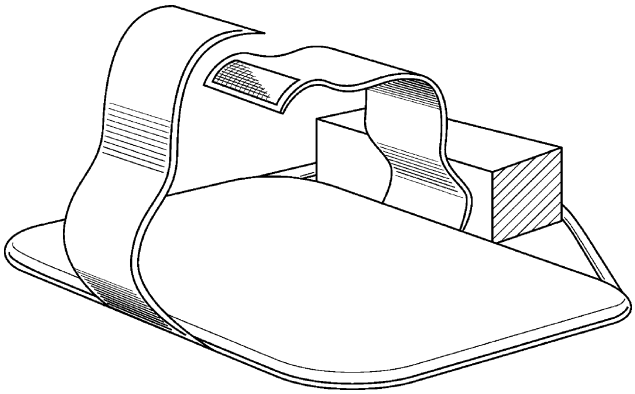

Fig. 1. Load distributing band compression system (AutoPulse technology) for cardiopulmonary resuscitation. Defibrillation can be performed during chest compression through the flat defibrillator electrodes (not shown) under the band. The electrocardiogram can be measured through the same electrodes.

The automated self-adjusting electromechanical device utilizes a load-distributing band to compress the anterior chest (Fig. 1). Manual CPR applies force to a small area over the sternum while A-CPR applies force over most of the anterior chest wall. Additionally, distributing compressive force over the anterior chest [3] may help to mitigate the severe chest wall trauma, abdominal injury and thoracic visceral injury that occur frequently during manual CPR [7–9]. Specifically, we sought to determine if A-CPR improves hemodynamics (peak aortic and coronary perfusion pressures) when compared to conventional CPR.

2. Methods

2.1. A-CPR chest compression device

The A-CPR device automatically adjusts to the size and shape of each patient. The device is constructed around a backboard that contains a motorized rotating shaft under microprocessor control (Fig. 1). The load distributing band is connected to the rotating shaft so that the band is tightened or loosened around the chest as the device operates. An anti-friction surface is incorporated to allow the band to slide freely around the subject. The microprocessor adjusts the band to the size of the subject being resuscitated and is programmed to provide a consistent 20% reduction in the anterior–posterior dimension of the subject's chest during the compression phase. The band distributes the compressive load over a large surface of the chest to reduce local stresses.

2.2. Protocol

The protocol compared the vascular pressures produced by A-CPR with those of manual CPR. With institutional review board approval, subjects were included following 10 min of failed standard advanced life support (ALS) protocol. This approach was designed to assess the vascular pressures produced by manual and A-CPR in the same patient without denying standard resuscitative measures initially. Terminally ill patients were enrolled from the intensive care units. Informed consent was not possible, which is ethically accepted for cardiopulmonary resuscitation research [10].

After 10 min of failed ALS, fluid-filled catheters were advanced into the thoracic aorta and the right atrium and placement was confirmed by pressure waveforms and chest radiograph. Following catheter placement, subjects received alternating periods of manual CPR and A-CPR for 90 s each. Manual and A-CPR chest compressions (treatment pairs) were cycled in each patient for as many repetitions as time would allow (1–6 pairs were recorded with a mean of three pairs per patient). All subjects were intubated and ventilated by bag-valve with room air. Manual chest compressions were administered without pauses for ventilation at 100 compressions/min. During manual CPR, ventilation was at a rate of 12 breaths/min between compressions. A-CPR was administered with five chest compressions to one ventilation at 60 compressions/min. The rate with A-CPR was limited to 60 min^{-1} to reduce power consumption, without compromising hemodynamics. Prior studies have shown that with compression for 40–50% of each cycle as occurs with A-CPR, hemodynamics are not dependent on rates from 60 to 120 min^{-1} [11]. Epinephrine (1 mg i.v. bolus) was given at the request of the attending physician at 3–5 min intervals. Defibrillation was performed as indicated. Spontaneous circulation was defined as being present if the systolic blood pressure was greater than 90 mmHg, and no CPR was being performed.

When death was officially declared, compressions were discontinued and a chest radiograph was taken to confirm catheter placement. The chest was palpated for evidence of flail chest or broken and dislocated ribs. In addition to catheter placement, the chest radiograph was examined for evidence of rib fractures.

2.3. Measurements

Vascular pressures were measured using the fluid filled catheters connected to Gould Statham pressure transducers. The force of compression was measured in a subset of patients. A load cell was placed under the back of the patient so that loads from manual and A-CPR compressions could be measured. The load cell was deformed by the static load of the patient's torso as well as the cyclic loading of chest compression. Pressure and force signals were digitized and stored by a microcomputer-based data acquisition system (National Instruments).

2.4. Statistical analysis

The objective of this crossover study was to compare A-CPR to manual CPR in generating vascular pressure rises

among cardiac arrest patients who failed to respond to at least 10 min of conventional resuscitative measures. The coronary perfusion pressure (CPP) was defined as the average difference between the aortic and right atrial pressure during mid to late chest decompression. Hemodynamic variables (peak and diastolic aortic, peak and diastolic right atrium, and coronary perfusion pressures) were evaluated for each treatment pair using the following method: each 90 s treatment period was divided into nine windows with 10 s duration. Blood pressure values were recorded in each successive 10 s window. Mean values were calculated from the nine windows giving the hemodynamic variables from that treatment period. The parameters reported are averages for all treatments in an individual subject.

Paired *t*-tests were used to determine whether A-CPR raised vascular pressures significantly higher than manual CPR. All statistical analyses were performed at the 5% significance level (two-sided) and were generated using SAS, version 6.12 [12]. Repeated measures of ANOVA were used to determine whether repeated treatment pairs in individual patients produced similar results, which allowed the reproducibility and stability of measurements to be assessed. Data were tested for normal distribution with the Shapiro–Wilk test. Data are presented as mean ± S.D.

3. Results

A total of 31 sequential subjects with in-hospital sudden cardiac arrest were screened between May 2000 to June 2001. Sixteen patients had useable aortic and right atrial pressure signals during manual and A-CPR, and hemodynamic data were reported and analyzed from those patients only. Demographic data are shown in Table 1. The average age was 68 ± 6 years, there were 11 males and the average weight was 73 ± 13 kg.

Table 1
Demographics ($n = 16$)

	Value	Min	Max
Age (years)	68 ± 6	61	78
Gender (% F)	31	–	–
Height (cm)	165 ± 10	150	185
Weight (kg)	73 ± 13	50	100
Chest depth (cm)	24 ± 3	20	31
Chest breadth (cm)	37 ± 5	30	46
Chest circumference (cm)	97 ± 11	86	121

The pre-arrest clinical status of each patient is shown in Table 2. These patients were terminally ill from heart disease and had additional co-morbidities. The average time between arrest and the start of the experimental protocol was 30 ± 18 min, ranging from 8 min (a second arrest) to 69 min. Only two patients required defibrillation, and the predominant rhythm noted at the onset of cardiac arrest was asystole.

When compared to manual CPR, one of the largest changes in pressures produced by A-CPR is shown in Fig. 2A. A-CPR markedly increased the peak and relaxation phase aortic pressure in this patient. During the compression-phase, the aortic and right atrial pressures are similar, therefore their difference is small. During the relaxation phase, however, the difference between the aortic and right atrial pressures (coronary perfusion pressure) generated by A-CPR is markedly higher than that generated by manual CPR. In contrast to Fig. 2A, Fig. 2B shows a patient where the change in relaxation pressures was near the mean value for all patients.

Table 2
Baseline clinical characteristics for each patient enrolled

ID	Time (min)	Arresting rhythm	Defibrillation attempt?	Clinical history
1	48	Asystole	Y	Chronic renal failure, hyperkalemia
2	11	Asystole	N	Unstable angina, AV block, pacemaker
3	8	Asystole	N	CAD, angioplasty, stent, CABG, unstable angina, pacemaker, renal failure
4	44	Asystole	N	CAD, diabetic
5	17	Bradycardia	N	CAD, valve disease
6	69	V. Fib.	Y	CAD, heart failure, emphysema
7	57	Bradycardia	N	Heart failure (R and L), CABG, external pacemaker
8	30	Asystole	N	CAD, stent, angioplasty, renal failure
9	11	Asystole	N	Stroke, SAH, endocarditis, mitral valve stenosis, atrial fibrillation
10	33	Asystole	N	CAD, aortic stenosis, pneumonia
11	47	Asystole	N	CAD, angioplasty
12	17	Asystole	N	CAD, acute renal failure, chronic hepatitis C
13	27	Asystole	N	ICD, ventricular arrhythmia, Chagas' disease
14	14	Bradycardia	N	Mitral regurgitation, ventricular cardiomyopathy, pulmonary infection, acute renal failure
15	20	Asystole	N	CAD, coma, chronic ventilator
16	38	Asystole	N	CAD, coma, chronic ventilator

Time is the duration of arrest prior to initiation of the experimental evaluation of A-CPR and manual CPR. Coronary artery disease (CAD), coronary artery bypass graft (CABG), sub-arachnoid hemorrhage (SAH), implantable cardioverter defibrillator (ICD).

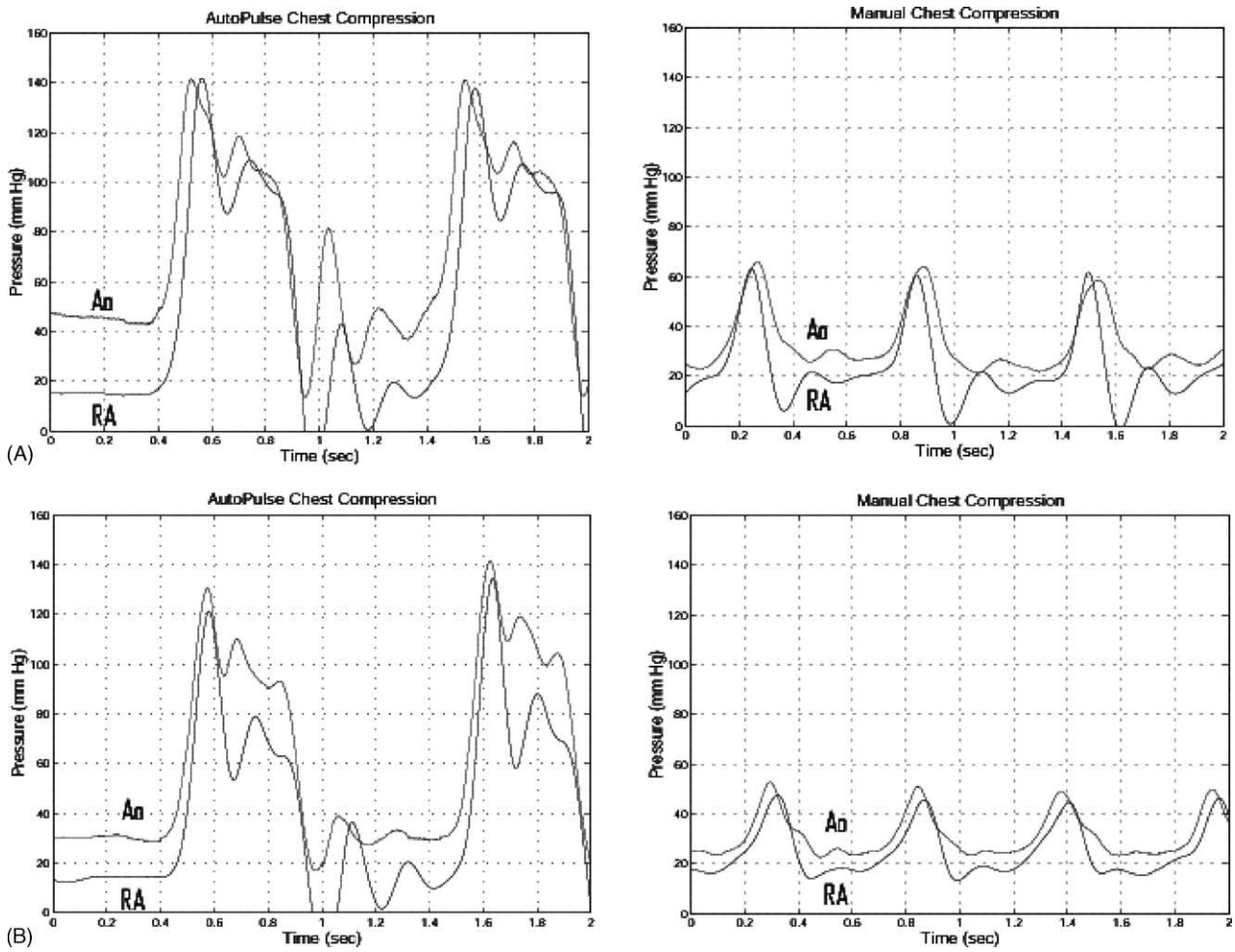

Fig. 2. Phasic vascular pressure traces during manual and A-CPR in two patients (A, B) reproduced from digital recordings. Record A shows one of the largest changes observed in aortic pressure and the diastolic aortic and right atrial pressure difference (coronary perfusion pressure) produced by A-CPR when compared to manual CPR. Record B shows a patient where the diastolic aortic and right atrium differences are near the mean values observed in this study.

Peak aortic pressures produced by manual CPR and A-CPR for individual patients are shown in Fig. 3, and coronary perfusion pressures for individual patients are shown in Fig. 4. For the group as a whole (Table 3), A-CPR increased peak aortic pressure (153 ± 28 mmHg versus 115 ± 42 mmHg, $P < 0.0001$), mean aortic pressure (70 ± 16 mmHg versus 56 ± 15 mmHg, $P < 0.0001$), and coronary perfusion pressure (20 ± 12 mmHg versus 15 ± 11 mmHg; $P < 0.015$; 95% CI: 8.7, 1.2). The results with paired *t*-tests can be considered valid since the data were normally distributed. In addition, the effects of A-CPR on the vascular pressures were not different when order was analyzed (i.e. A-CPR first or second). These pressures may not be representative of the 15 patients where useable pressure signals were not obtained.

The force of compression was measured in a subset ($n = 10$) of patients (Table 3). Peak force with manual compressions was 51 ± 20 kg and with A-CPR compressions peak force was 125 ± 18 kg ($P < 0.0001$). The pressure applied to the chest was calculated to be 203 ± 20 mmHg with

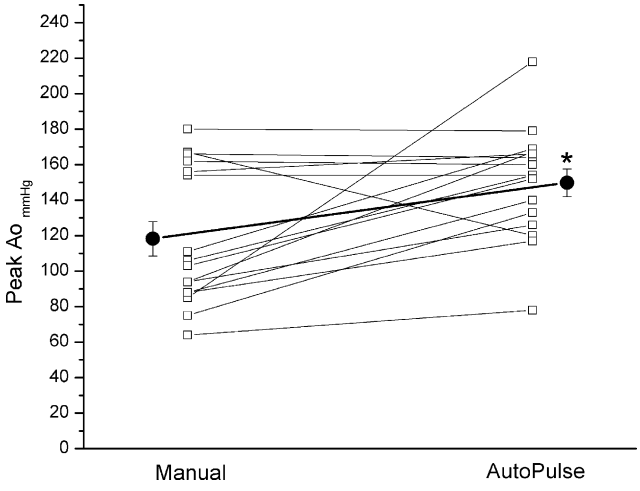

Fig. 3. Peak aortic pressure during manual and A-CPR. Each connected pair of squares are data from one patient. Symbols to the left and right of the pressures are means ± S.E. There is a significant increase in aortic pressures with A-CPR (* $P < 0.0001$).

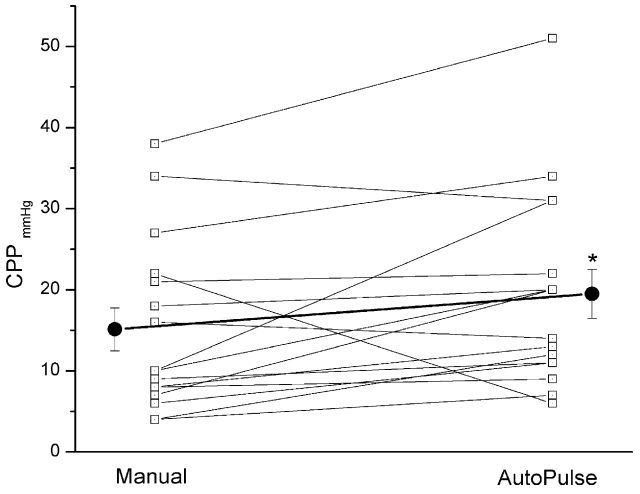

Fig. 4. Coronary perfusion pressure during manual and A-CPR. Each connected pair of squares are data from one patient. Symbols to the left and right of the pressures are means ± S.E. There is a significant increase in vascular pressures with A-CPR ($^*P < 0.015$).

A-CPR while the pressure with manual CPR was estimated as 1381 ± 432 mmHg. The increased pressure with manual CPR is due to the much smaller area of application of force with manual CPR compared with A-CPR.

Only 1 subject of the 16 analyzed showed a substantial decrease in pressures with A-CPR when compared to manual CPR. Subject #5 received two cycles of chest compression, A-CPR initially in the first treatment pair and manual chest compressions initially in the second treatment pair. The first pair showed lower CPP (6 mmHg with A-CPR versus 26 mmHg with manual CPR) and peak aortic pressure (121 mmHg with A-CPR versus 183 mmHg with manual CPR). The second pair showed the same pattern with lower CPP (6 mmHg with A-CPR versus 19 mmHg with manual CPR) and lower aortic pressure (118 mmHg with A-CPR versus 151 mmHg with manual CPR). Epinephrine (1 mg i.v.) was administered simultaneously with the initiation of both manual chest compression periods. The force delivered to the patient's chest was higher than average with manual chest compression (82 kg for patient #5 versus an average of 51 ± 16 kg) while the A-CPR device delivered force was lower than average (100 kg for patient #5 versus an average of 125 ± 12 kg). Further data collection was not attempted as it was observed that the A-CPR device's motor current was exceedingly high (compared to normal operation) and may have indicated a motor failure.

Table 3
Hemodynamics and force from A-CPR and manual CPR

	Average	Min	Max	*P* (vs. manual)
Aortic peak				
A-CPR	153 ± 28	78	255	<0.0001
Manual CPR	115 ± 42	45	180	
Aortic diastolic				
A-CPR	29 ± 12	8	58	0.3660
Manual CPR	27 ± 10	8	49	
Aortic mean				
A-CPR	70 ± 15	45	106	<0.0001
Manual CPR	56 ± 16	31	90	
Right atrial peak				
A-CPR	129 ± 32	82	184	<0.0001
Manual CPR	83 ± 40	23	172	
Right atrial diastolic				
A-CPR	11 ± 7	0	22	0.6571
Manual CPR	12 ± 6	3	24	
Right atrial mean				
A-CPR	50 ± 12	35	74	<0.0001
Manual CPR	36 ± 13	14	68	
CPP				
A-CPR	20 ± 12	7	51	0.0150
Manual CPR	15 ± 11	3	38	
Compressive force (kg)				
A-CPR	125 ± 18			<0.0001
Manual CPR	51 ± 20			

Pressures are in mmHg. A-CPR is AutoPulse CPR.

One patient evaluated for the present study but not included in the 16 reported with hemodynamics above had spontaneous return of circulation while receiving A-CPR. The patient had been treated with 11 min of failed ALS for an asystolic arrest following myocardial infarction. During that time, femoral catheters were placed for the study, epinephrine was given four times (1 mg i.v.) and atropine was given twice (1 mg i.v.). All of these efforts failed to restart circulation and the patient was randomized to receive A-CPR first. The pressures generated by manual compressions during the initial ALS period were not recorded in this subject. The CPP immediately preceding the start of AutoPulse function was 4 mmHg. Within 30 s of the device's activation, spontaneous circulation was noted. Pressures immediately before the spontaneous cardioversion were CPP = 28 mmHg, peak Ao = 200 mmHg, peak RA = 180 mmHg. The patient stabilized to a heart rate of 120 beats/min and a blood pressure of 170/60 mmHg. The patient survived over 1 h until he succumbed to a second cardiac arrest.

4. Discussion

A number of studies have shown that a major factor affecting survival from cardiac arrest is the level of vascular pressures, especially the coronary perfusion pressure, generated during resuscitation [2–4]. This is probably most applicable after failure of initial defibrillation or when defibrillation is not indicated. Recent evidence suggests that circulation may be an important component of resuscitation for patients with ventricular fibrillation as well [5,6]. Invasive techniques such as open-chest cardiac massage [13] and cardiopulmonary bypass [14] increase vascular pressures compared with manual CPR, but have not as yet come into widespread pre-hospital use in part because they must be performed by physicians. Non-invasive techniques could have more general use, but

methods such as chest compression with simultaneous ventilation [15,16], and manual chest compression at increased rates [17,18], have generated pressures only marginally different from those produced by manual CPR. Newer promising techniques, which require skilled users, include manual chest compression with active decompression [19,20], and the use of a valve to impede airflow during chest decompression to increase negative intrathoracic pressure [20,21]. Circumferential thoracic vest inflation techniques can improve pressures substantially [3,22], but the size, weight, and power requirements of the vest system make it logistically difficult to use.

We tested a novel chest compression system based on a load distributing band that produces anterior–posterior chest compression. The A-CPR device applies force over a much larger portion of the chest than occurs with manual CPR, covers a similar portion of the anterior chest as vest CPR, but uses much less power than vest CPR. Increased force can be applied to the chest generating large changes in vascular pressures (Fig. 2) while reducing the pressure on the chest itself, thus reducing the likelihood of trauma by decreasing the local stresses.

We studied the hemodynamics produced by A-CPR and manual CPR in a situation where there was minimal risk to the subject. We chose the end of standard resuscitative measures as an appropriate time to test A-CPR. A-CPR increased vascular pressures compared with manual CPR (Figs. 3 and 4, Table 3).

The peak aortic pressures reported for manual CPR (Table 3) in this study were at least as high as those reported by other investigators [23], as are the coronary perfusion pressures [4,23]. It is very unlikely, therefore, that inadequate compression force was responsible for the observation that manual CPR produced lower pressures than A-CPR (Table 3, Figs. 3 and 4). In fact, the manual compression forces used in this study were likely to be higher than those typically used during CPR [3,11], especially in the one patient where the pressures were increased with manual CPR.

The coronary perfusion pressures produced by A-CPR might be even higher if the A-CPR device was applied earlier in cardiac arrest. Laboratory studies of resuscitation showed that coronary perfusion pressures during manual CPR decreased substantially toward the end of a 20 min period of resuscitation [3], likely to be the result of decreased peripheral resistance causing increased arterial runoff. This reduced peripheral resistance is probably the result of decreased arterial tonus caused by direct damage to the vascular smooth muscle or by changes in smooth muscle cell receptors.

5. Conclusion

In a terminally ill patient population, A-CPR demonstrated a clinically significant improvement in hemodynamics including coronary perfusion pressure when compared to high force manual chest compressions. If spontaneous circulation is restored early in cardiac arrest before irreversible organ damage has occurred, then long-term survival may be possible. Additional human studies are indicated.

Acknowledgements and Conflict of Interest

Dr. Halperin has a financial interest in Revivant Corporation. All financial interests are governed by policies of the Johns Hopkins University as implemented by the Johns Hopkins University Advisory Committee on Conflicts of Interest. Dr. Halperin had no role in enrolling patients or in analyzing the raw data. Supported by a grant from Revivant Corporation, Sunnyvale CA, to the InCor.

References

[1] Zheng ZJ, Croft JB, Giles WH, Mensah GA. Sudden cardiac death in the United States, 1989 to 1998. Circulation 2001;104(18):2158–63.
[2] Ralston SH, Voorhees WD, Babbs CF. Intrapulmonary epinephrine during prolonged cardiopulmonary resuscitation: improved regional blood flow and resuscitation in dogs. Ann Emerg Med 1984;13(2):79–86.
[3] Halperin HR, Guerci AD, Chandra N, et al. Vest inflation without simultaneous ventilation during cardiac arrest in dogs: improved survival from prolonged cardiopulmonary resuscitation. Circulation 1986;74(6):1407–15.
[4] Paradis NA, Martin GB, Rivers EP, et al. Coronary perfusion pressure and the return of spontaneous circulation in human cardiopulmonary resuscitation. JAMA 1990;263(8):1106–13.
[5] Cobb LA, Fahrenbruch CE, Walsh TR, et al. Influence of cardiopulmonary resuscitation prior to defibrillation in patients with out-of-hospital ventricular fibrillation. JAMA 1999;281(13):1182–8.
[6] Wik L, Hansen TB, Fylling F, et al. Delaying defibrillation to give basic cardiopulmonary resuscitation to patients with out-of-hospital ventricular fibrillation: a randomized trial. JAMA 2003;289(11):1389–95.
[7] Silberberg B, Rachmaninoff N. Complications following external cardiac massage. Surg Gynecol Obstet 1964;111:6–10.
[8] Wolfe WG, Dudley AW, Wallace AG. A pathologic study of unsuccessful cardiac resuscitation. Arch Surg 1968;96:123–6.
[9] Nadel EL, Fine EG, Krischer JP, Davis JH. Complications of CPR. Crit Care 1981;9(5):424.
[10] Hook CC, Koch KA. Ethics of resuscitation. Crit Care Clin 1996;12(1):135–48.
[11] Halperin HR, Tsitlik JE, Guerci AD, et al. Determinants of blood flow to vital organs during cardiopulmonary resuscitation in dogs. Circulation 1986;73(3):539–50.
[12] SAS Procedures, SAS Procedures Guide. Cary, NC: SAS Institute Inc.; 1990.
[13] Redding JS, Cozine R. A comparison of open-chest and closed-chest cardiac massage in dogs. Anesthesiology 1961;22:280.
[14] Reich H, Angelos M, Safar P, Sterz F, Leonov Y. Cardiac resuscitability with cardiopulmonary bypass after increasing ventricular fibrillation times in dogs. Ann Emerg Med 1990;19(8):887–90.
[15] Chandra N, Rudikoff M, Weisfeldt ML. Simultaneous chest compression and ventilation at high airway pressure during cardiopulmonary resuscitation. Lancet 1980;1(8161):175–8.
[16] Koehler RC, Chandra N, Guerci AD, et al. Augmentation of cerebral perfusion by simultaneous chest compression and lung inflation with abdominal binding after cardiac arrest in dogs. Circulation 1983;67(2):266–75.

[17] Maier GW, Tyson Jr GS, Olsen CO, et al. The physiology of external cardiac massage: high-impulse cardiopulmonary resuscitation. Circulation 1984;70(1):86–101.
[18] Feneley MP, Maier GW, Kern KB, et al. Influence of compression rate on initial success of resuscitation and 24 hour survival after prolonged manual cardiopulmonary resuscitation in dogs. Circulation 1988;77(1):240–50.
[19] Lurie KG, Lindo C, Chin J. CPR: the P stands for plumber's helper. JAMA 1990;264(13):1661.
[20] Plaisance P, Lurie KG, Payen D. Inspiratory impedance during active compression–decompression cardiopulmonary resuscitation: a randomized evaluation in patients in cardiac arrest. Circulation 2000;101(9):989–94.
[21] Lurie KG, Zielinski T, McKnite S, Aufderheide T, Voelckel W. Use of an inspiratory impedance valve improves neurologically intact survival in a porcine model of ventricular fibrillation. Circulation 2002;105(1):124–9.
[22] Halperin HR, Tsitlik JE, Gelfand M, et al. A preliminary study of cardiopulmonary resuscitation by circumferential compression of the chest with use of a pneumatic vest. N Engl J Med 1993;329(11):762–8.
[23] Swenson RD, Weaver WD, Niskanen RA, Martin J, Dahlberg S. Hemodynamics in humans during conventional and experimental methods of cardiopulmonary resuscitation. Circulation 1988;78(3):630–9.